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THE ASSOCIATION BETWEEN CARDIOVASCULAR RISK FACTORS AND CARDIOVASCULAR FITNESS

by

(C) MARGARET FRANCES KAVANAGH

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

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DEDICATION

TO MY PARENTS

ABSTRACT

The objective of this investigation was to examine the relationship between various known risk factors for coronary heart disease (CHD) and an individual's level of fitness as determined by a submaximal exercise test at a community/university fitness unit. The risk factors that were investigated included hyperlipidemia, hypertension, cigarette smoking, diabetes mellitus, family history, physical activity, obesity and alcohol consumption. The subjects were adult, Canadian males and females who voluntarily presented themselves for fitness appraisal at the University of Alberta fitness unit during the time period of December 1983 to November 1984. This fitness unit was open to the general public for individual fitness and lifestyle appraisal. The fitness test was based upon the Standardized Test of Fitness plus a submaximal bicycle ergometer test to assess cardiovascular fitness. The subjects were divided into low fit (<5—30% ile) and high fit (70—>95% ile) categories based on the classification of their predicted VO_2 MAX in the Swedish norms. The low fit sample demonstrated significantly higher serum triglycerides, serum cholesterols, lipid ratios, fasting blood sugars, resting systolic and diastolic blood pressures, body weight and percent body fat, and alcohol consumption as compared to the high fit sample. The low fit sample also had a greater likelihood of having a positive family history for CHD or diabetes mellitus, and lower high density lipoproteins and lower activity levels. The unfit subjects also had a significantly higher risk, based on a multiple risk assessment, of developing CHD within 6 years when compared to the high fit subjects. Many of these abnormalities are related to an unhealthy lifestyle. It behooves the fitness appraiser/counsellor to be aware of these problems and to advise their clients appropriately, for the apparently normal healthy population seeking fitness appraisal is not completely free of risk.

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Table of Contents

Chapter		Page
I.	INTRODUCTION	1
	PURPOSE	3
	LIMITATIONS	3
	DELIMITATIONS	3
	DEFINITION OF TERMS	3
II.	REVIEW OF LITERATURE	5
	INTRODUCTION	5
	AGE AND SEX	5
	SERUM LIPIDS	6
	HYPERTENSION	11
	CIGARETTE SMOKING	14
	EXERCISE	16
	FAMILY HISTORY	18
	DIABETES MELLITUS	19
	OBESITY	20
	ALCOHOL	22
	MULTIPLE RISK	23
	SUMMARY	24
III.	METHODOLOGY	25
	SUBJECTS	25
	FITNESS UNIT PROCEDURES	25
	HOSPITAL PROCEDURES	26
	STATISTICAL ANALYSIS	29
IV.	RESULTS AND DISCUSSION	31
	AGE AND SEX	32
	SERUM LIPIDS	34

HYPERTENSION 37
CIGARETTE SMOKING 39
EXERCISE 41
FAMILY HISTORY 43
DIABETES MELLITUS 45
OBESITY 45
ALCOHOL 48
MULTIPLE RISK 49
SUMMARY OF RISK FACTOR DATA 50
LIMITATIONS 53
V. CONCLUSIONS 58
BIBLIOGRAPHY 60
APPENDIX A: PAR-Q AND CONSENT FORM 71
APPENDIX B: FITNESS UNIT DATA SHEET 72
APPENDIX C: HEALTH HAZARD APPRAISAL QUESTIONNAIRE 73
APPENDIX D: ASTRAND-RHYMING NOMOGRAM 75
APPENDIX E: AGE CORRECTION FACTOR FOR ASTRAND-RHYMING NOMOGRAM 76
APPENDIX F: SWEDISH NORMS FOR CLASSIFICATION OF VO_2 MAX. 77

List of Tables

Table 1: Diastolic Blood Pressure 13

Table 2: Body Weight as a Risk Factor (Stamler, 1979) 22

Table 3: Cardiovascular Fitness Criteria 26

Table 4: Hypertensive Blood Pressure Criteria 27

Table 5: Blood Lipid and Glucose Limits 28

Table 6: Obesity Criteria 28

Table 7: Classification of Physical Activity 29

Table 8: Subjects Tested vs. Fitness Classification 31

Table 9: Percentage of Subjects Tested at U of A Stress Testing Lab with their Mean Ages and Standard Deviations 32

Table 10: Means and Standard Deviations for Blood Parameters 35

Table 11: Means and Standard Deviations for Blood Pressure 37

Table 12: Subjects with Elevated Blood Pressure 38

Table 13: Smoking History by Sex 40

Table 14: Smoking History by Fitness Classification 40

Table 15: Exercise Level by Sex 42

Table 16: Exercise Level by Fitness Classification 42

Table 17: Subjects with a Positive Family History 44

Table 18: Anthropometric Data 46

Table 19: Number of Drinks per Day 48

Table 20: Means and Standard Deviations for Probability (per 100) of Developing CHD in 6 Years 49

Table 21: Regression Equations and Correlation 55

LIST OF SYMBOLS

VO_2MAX	—	Maximum oxygen uptake
LDL	—	Low density lipoprotein
VLDL	—	Very low density lipoprotein
HDL	—	High density lipoprotein
SBP	—	Systolic blood pressure
DBP	—	Diastolic blood pressure
CHD	—	Coronary heart disease

Chapter I

INTRODUCTION

Coronary heart disease (CHD) is a major cause of death in the U.S. and Canada (Statistics Canada, 1983a; Atherosclerosis Study Group, 1984). The death rates in Canada for men are 239/100,000 of the population and 161/100,000 of the population for women (Statistics Canada, 1983b). The incidence and prevalence is directly related to hereditary factors and the lifestyle that predominates in the civilized world (Atherosclerosis Study Group, 1984).

There are specific risk factors which have been identified as having a direct relationship to the development of CHD. These risk factors include hypertension, hyperlipidemia, cigarette smoking, obesity, diabetes mellitus, lack of physical activity, alcohol intake and family history (Dawber, 1980; Kaplan and Stamler, 1983; Kannel et al., 1984; Atherosclerosis Study Group, 1984). The relationship of these risk factors to CHD appears to be cause and effect in some cases, yet more tenuous in others.

One such relationship is that of physical activity to CHD. Physical activity has been shown to be inversely related to the incidence and prevalence of CHD (ie. the more active one is the lower the incidence of CHD) (Morris et al., 1966; Paffenbarger and Hale, 1975; Paffenbarger et al., 1978b; Fox et al. 1981, Eichner, 1983). In addition, physical activity and higher fitness levels have been linked to reduced risk factors for CHD (Lopez et al., 1974; Hickey et al., 1975; Cooper et al. 1976; Wyndham, 1979; Erickssen et al., 1981; Paffenbarger et al., 1983).

In recent years there has been an increased interest in physical activity and fitness within the general population. The reasons for this renewed interest in fitness are multifactorial with the health benefits of physical activity being an important factor (Fitness and Amateur Sport, 1983). Due to this increase in attention to physical activity many people are seeking the advice of fitness appraisers and physical educators. Fitness testing centres can take many forms from the community based unit, with persons trained in fitness testing techniques but not trained to handle medical problems and clinical evaluations, to formal medical stress testing

labs equipped to diagnose heart disease. Stress testing labs are equipped to manage subject's with previous medical problems, such as hypertension or diabetes. The community units are not equipped to handle such problems. Stress testing labs are aware of the types of patients that they are requested to evaluate through physician referrals. These patients present themselves at units with previous medical problems and recognized risk factors for heart disease; for example, they often have had symptoms of chest pain that were suspicious of cardiac disease. In that respect the staff of the stress testing lab are aware of the problems that they may encounter.

Persons presenting themselves at community fitness units are required to be healthy and symptom-free. They are to be free of any medical problems that may result in untoward difficulties during the testing procedures. However, is this really the case? Are so called apparently normal subjects, really free of risk factors and potential problems? Are there any differences in the CHD risk factors in the fit versus the unfit subjects who present themselves at community fitness units? If there are differences in the risk factors within this population, then fitness appraisers should be made aware of these for safety and counselling purposes.

Previous work in this area of fitness evaluation and risk factors does not completely answer these questions. Cooper et al. (1976) studied CHD risk factors and fitness levels in males only but they did not have to be completely symptom free for their unit was capable of managing and diagnosing cardiac complications. Hickey et al. (1975) again studied men only but he was interested in CHD risk factors and leisure activity and not actual fitness testing situations. Erickssen et al. (1981) studied males in a testing situation but the CHD risks factors evaluated were deficient. Also his subjects were recruited and did not voluntarily seek fitness assessment and advice. Gibbons et al. (1983) studied risk factors in women with respect to fitness. This was the most complete work because it looked at many different risk factors, including the lipoprotein fractions, but other risk factors such as family history were not examined. Therefore, the present discussion will attempt to answer the above questions more thoroughly.

PURPOSE

The purpose of this study is to investigate the relationship between the levels of fitness, as determined in a University or community based fitness testing facility, to the risk factors for coronary heart disease in adult Canadians.

LIMITATIONS

a) The subjects were chosen from the adult population of Edmonton who voluntarily presented themselves to the fitness unit for fitness assessement.

b) The subjects who returned for further testing to the university hospital stress lab did so voluntarily.

c) All subjects were encouraged strongly to return for the appropriate blood work but again this was done on a volunteer basis.

DELIMITATIONS

a) All subjects must be PAR-Q negative (Chisholm, 1978).

b) The data was collected during the time period of December 15, 1983 to November 30, 1984.

c) Only the low fitness (<5—30% ile) and the high fitness (70—>95% ile) groups of subjects from the cardiovascular test of fitness, were chosen.

d) Only the following risk factors were considered for evaluation: hyperlipidemia, hypertension, cigarette smoking, physical activity, hyperglycemia, obesity, family history and alcohol intake.

DEFINITION OF TERMS

Risk factor - "A trait that places an individual at risk for cardiovascular disease. This concept is derived from a variety of epidemiological studies on cardiovascular mortality rates among countries, racial, occupational and religious sub-groups, and most importantly over a

long period of time." (Kannel et al., 1984).

Hypercholesterolemia - Elevated serum cholesterol or more specifically a serum cholesterol level >6.5 mmol/l (250 mg/dl).

Hypertriglyceridemia - Elevated serum triglycerides or more specifically a serum triglyceride level >2.0 mmol/l (77 mg/dl).

Hypertension - Elevated arterial blood pressure meeting the following criteria:

a) Systolic blood pressure > 140 mmHg and/or

b) Diastolic blood pressure > 90 mmHg

Atherosclerosis - Lipid deposits in the subinternal layer of the larger arteries leading to narrowing of the vessels.

Coronary Heart Disease (CHD) - Atherosclerosis of the coronary arteries which leads to ischemia and infarction of cardiac muscle.

Incidence - The member of new cases developing within a given period of time (Dawber, 1980).

Prevalence - Amount of disease existing at a particular time (Dawber, 1980).

Chapter II

REVIEW OF LITERATURE

INTRODUCTION

The cause of coronary heart desease (CHD) is multifactorial in nature. There are major and minor risk factors which are related to the development of CHD. Major risk factors are modifiable factors that display a cause and effect relationship with CHD (Pooling Project, 1978; Stamler, 1979). The major risk fators include hypercholesterolemia, hypertension and cigarette smoking (Stamler, 1979).

Minor risk factors show some relationship to CHD but the association is not as strong, or can not be modified through lifestyle changes. The minor risk factors include diabetes mellitus, obesity sedentary lifestyle, alcohol and family history (Stamler, 1979). In addition, age and sex play a major role but there is little one can do to alter these factors (Dawber, 1980).

The data will show "that the relationship between the major risk factors and susceptibility to CHD are consistent, strong, graded and independant, and that they meet, the test of temporal sequence (i.e. presence of the trait precedes that of the event in time), but that they also hold up excellently when put to the further test of predictive ability" (Pooling Project, 1978).

Each of these risk factors will be examined to expand upon their relationship to CHD. By so doing the basis for the current investigation will be demonstrated and the link to fitness will be uncovered.

AGE AND SEX

Age and sex are two vitally important risk factors but there is nothing one can do to alter these factors. Coronary heart disease (CHD) is known to occur much more frequently in men than women (Dawber, 1980). CHD is a male dominant disease and it occurs at a much

younger age in men than women particularly before the age of 60. Men are 10 times more likely than women to have a myocardial infarction before the age of 50, and the overall risk of developing CHD is 2 times greater in men than women (Dawber, 1980). In addition, women lag behind men in the incidence of CHD by 10 years, but this gap narrows with advancing age At any level of risk factors singly, or in combination, women have a distinct advantage over men (Kannel et al., 1984). According to the Framingham study, the overall risk of developing CHD by age 65 was 37% for men and 18% for women (Kannel et al., 1976).

Angiographic evidence of symptom-free CHD is also present in males with additional risk factors, but not in females (Cramer et al., 1966). In addition, risk factors HDL cholesterol, LDL cholesterol, and total cholesterol are less atherogenic in females before age 50 (Kaplan and Stamler,1983).

The incidence and prevalence of CHD increases with age (Pooling Project, 1978; Dawber, 1980). The Pooling Project (1978) reported an increased incidence of CHD with age. For men age 40—44 the incidence rate was 15.5/1000 men and for men age 60—64 the incidence rate was 95.2/1000 men. Bruce et al. (1983) reported a significant increase in risk over the age of 55. Not only is age a risk factor, but it acts independently of other risk factors (Uhl, et al. 1981).

SERUM LIPIDS

Lipid analysis is made up of several parts which include total serum cholesterol, serum triglycerides, low density lipoproteins (LDL), very low density lipoproteins (VLDL), high density lipoproteins (HDL) and ratios of the lipoprotein fractions. Initial research only included triglycerides and total cholesterol but the advent of lipid fractionation has further increased our knowledge. The VLDL fraction carries triglycerides predominately, and LDL and HDL carry the cholesterol fractions. Specifically, HDL transports approximately 20% of the total serum cholesterol and LDL transports 50—66% of the total serum cholesterol (Castelli et al., 1977; Kaplan and Stamler, 1983).

It has been postulated that the LDL fraction is responsible for carrying cholesterol to the periphery, and thus making it available for atherogenesis; and the HDL fraction transports cholesterol from the periphery to the liver for degradation and disposal (Miller and Miller, 1975, AHA, 1984). This mechanism may account for the atherogenic influence of LDL cholesterol and the protective influence of HDL cholesterol; for if a larger part of the total cholesterol is sequestered in the HDL form, then smaller amounts remain for the atherogenic LDL form (Castelli et al., 1977; Gordon et al., 1977). Accumulation of cholesterol in atheroma occurs when excess cholesterol remains due to an imbalance of synthesis and removal. Atherosclerosis is not only accelerated by an increase in cholesterol synthesis, but also a decrease in its disposal (Miller and Miller, 1975). A normal total serum cholesterol does not exclude elevation of the atherogenic fractions yet keeping the total within the normal range (Rossner, 1982).

Considerable evidence has accumulated indicating that in addition to total serum cholesterol the manner in which the cholesterol is distributed or transported is associated with a high risk of CHD (Uhl et al., 1981). There is no doubt that total serum cholesterol is related to the development ot CHD (Kannel et al., 1984). Not only is there a relationship between CHD and total cholesterol but it is dose related, occurs in both sexes, precedes the disease and is independant of other risk factors; thus satisfying the criteria for a major risk factor. A strong relationship between serum cholesterol and the incidence of CHD was discovered in the seven countries data (Keys, 1970). In countries like the U.S. where the average serum cholesterol level is high (250 mg%), there is a high incidence of CHD; and in Japan where the average serum cholesterol level is low (180 mg%), the incidence of CHD is low. This same relationship holds for the dietary fats (Keys, 1970).

A total serum cholesterol exceeding 200 mg/dl (5.2 mmol/l) is incompatible with optimal cardiovascular health (Kannel et al., 1984; AHA, 1984). However, the precise level at which the risk increases dramatically is controversial. The Framingham study (Dawber, 1980) reported an increased risk for young men with total cholesterol levels >260 mg% (6.76 mmol/l)

of 4 times that of men with levels <200 mg% (5.2 mmol/l).

The relative risk from elevated serum cholesterol is greater in the young than in older persons. The impact of total serum cholesterol wanes after age 55 such that it no longer is predictive of CHD (Kannel et al., 1984). The risk increases 5 fold over the range of serum cholesterol which is considered within normal limits. However, the greatest risk appears to occur above the 250—260 mg/dl (6.5—6.76 mmol/l) range.

The risk of a first time event for men with serum cholesterol levels >240 mg/dl (6.24 mmol/l) was 2—2.4 times that of men with values of <218 mg/dl (5.7 mmol/l) (Castelli et al., 1977). On average men suffering from CHD have an increase in their total serum cholesterol of 6 mg/dl (0.16 mmol/l) over their healthy counterparts, and an increase of 21 mg/dl (0.55 mmol/l) in triglycerides (Castelli et al,1977). Finally, the U.S. railroad workers (Keys, 1970) had a prevalence of CHD 2.48 times greater when the cholesterol level was >260 mg % (6.76 mmol/l) as opposed to <230 mg % (6.0 mmol/l).

Since LDL cholesterol is the lipoprotien fraction that carries, the majority of the atherogenic serum cholesterol, a positive relationship between LDL cholesterol and CHD would be expected. This positive relationship between total cholesterol and LDL cholesterol to CHD has been documented (Gordon et al.,1977; Dawber,1980).

Recently, a new theory about the link between serum cholesterol, low density lipoprotein and atherosclerosis has been proposed (Goldstein et al., 1983; Brown and Goldstein, 1984). This theory suggests that elevated serum cholesterol and LDL results from a decrease in either the number, or the sensitivity of the LDL receptors. LDL receptors are present on all cells but are especially prominent on liver cells. These receptors bind with circulating LDL and this leads to cellular uptake through endocytosis and ultimately lysosomal degradation. If the LDL receptors are absent or insensitive to circulating LDL, then there will be a build-up of LDL in the bloodstream. In the case of familial hypercholesterolemia there is a total absence of receptors in the homozygotes and a 50% decrease in the number of receptors in the heterozygotes. This results in a decrease in the removal rate of LDL from the circulation

by two thirds and by one third respectively, and these persons generally develop symptomatic atherosclerosis by age 20 and 40 respectively. The vast majority of persons with elevated serum LDL do not have familial hypercholesterolemia. Genetic and epidemiologic studies suggest that they do have subtle abnormalities in many genes whose deleterious effects are aggravated by environmental factors such as high intake of dietary fat, calories and cholesterol. The problem here is thought to result from feedback suppression of the hepatic LDL receptors, thus encouraging the LDL cholesterol to accumulate in the bloodstream.

It appears that the critical value for total serum cholesterol is in the 240—260 mg/dl range (6.24—6.76 mmol/l). The risk of developing CHD increases significantly in this range, and it is for this reason that the value of 250 mg/dl (6.5 mmol/l) was selected as the critical value for determining elevated serum cholesterol, regardless of age.

Serum triglycerides have been positively linked to CHD, however not independantly (Gordon et al., 1977; Rossner, 1982; AHA, 1984; Kannel et al., 1984). They are related to diabetes mellitus, obesity and alcohol consumption; all of which are risk factors for CHD. Castelli et al.(1977) also found a positive relationship between CHD and triglyceride levels. A statistically significant relationship for the prevalence of CHD was found between triglycerides or LDL cholesterol when the other was held constant. A strong relationship has been noted in women between serum triglycerides and CHD, but again this relationship is not independant. However, Gordon et al. (1977) did find a strong relationship between triglycerides and diabetes mellitus. Further discussion on this topic will be presented in the diabetes section.

An inverse relationship between HDL cholesterol and CHD has been found (Castelli et al., 1977; Gordon et al., 1977). Persons with HDL levels below 35 mg/dl (0.91 mmol/l) have an incidence rate for CHD 8 times that of persons with HDL levels > 65 mg/dl (1.7 mmo/l) (Castelli et al., 1977). Women tend to have on average HDL levels that are 10 mg/dl higher than age matched males; and perhaps this gives women more of a protective advantage over men (Castelli et al., 1977). Based on this data the value of 0.9 mmol/l was chosen as the demarcation point between normal and low values of HDL in this study.

In vitro studies have demonstrated an efflux of cholesterol from atheromatous tissue by the addition of HDL to the incubation medium (Miller and Miller, 1975). This supports the theory that HDL helps to transport cholesterol away from the cellular level to the liver for disposal.

Some authors suggest that it is not the values of VLDL, LDL and HDL cholesterol but rather the ratios of the lipid fractions that are important (Wilson et al., 1980; Rossner, 1982). For example, Uhl et al. (1981) demonstrated that an elevated total cholesterol to HDL ratio was highly predicitve of CHD, in fact much more so than HDL or total cholesterol alone. They found the predictive value for CHD of a total cholesterol/HDL ratio $>$ 6.0 to be 64% and when $<$ 6.0 to be only 2%. In addition, this ratio relationship to CHD appears to be independant of age.

The impact of serum cholesterol as a predictor of CHD wanes after age 55 but the LDL/HDL ratio continues its predictive powers (Kannel et al., 1984). The Framingham study (Kannel et al., 1984) demonstrated increased risk when the LDL/HDL ratio was greater than 3.6 and when the total cholestero/HDL ratio was greater than 5.0.

The ratio available in the U of A hospital laboratory is the LDL + VLDL/ HDL ratio. This takes into account all of the atherogenic cholesterol and any potential effect of the triglycerides as well. The upper limit of normal in the hospital laboratory is 4.0 and this value will be used in this study as well.

Numerous lipid intervention programmes to lower serum lipids have reinforced the importance of lipids to the development of CHD. The Lipids Research Clinics Program (1984) produced a 25% decrease in total cholesterol and a 35% decrease in LDL cholesterol resulting in a 50% decrease in the incidence of CHD. Significant reduction was attained by using diet alone but an additional decrease in cholesterol was attained by the use of cholestyramine, a bile acid sequestor.

In Finland, where the highest incidence and prevalence of CHD exists, serum cholesterol was lowered by 3% with a specific treatment regimes. Accompanying changes in

cigarette smoking and blood pressure resulted in lowering of the CHD mortality rate when compared to the rest of the country (Puska et al., 1983).

HYPERTENSION

There is a definite, well defined relationship between elevated blood pressure, both systolic and diastolic to increased risk of developing CHD (Kannel, 1975; Kannel et al., 1980 a and b). The Framingham data (Dawber, 1980; Kannel et al., 1980 a and b; Kannel et al., 1984) demonstrated this relationship quite clearly. Using casual blood pressure readings, as opposed to a predetermined protocol including a rest period before taking blood pressure, they found a definite positive relationship between systolic and diastolic blood pressure and CHD. They also noted a marked increase in risk for cerebrovascular accidents with hypertension. The Framingham group utilized the following for classifying the blood pressure readings: normal (<140/95 mmHg), borderline (140/90 mmHg—160/95 mmHg) and elevated (both >160/95 mmHg). Among patients who died from CHD 37% of men and 51% of women had distinct hypertension (>160/95 mmHg). However, if you include the borderline group (>140/90 mmHg) then the mortality statistics increase to 73% for men and 81% for women (Kannel, 1975). This points out that even borderline elevations in blood pressure have an increased risk associated with them.

The difficulty with hypertension is defining a demarcation point above which blood pressure is considered elevated. In fact blood pressure is more of a continuum, and there is not a clear cut level above which it is elevated and below which it is not. Despite this, there are levels above and below which the risk of CHD is markedly elevated. The World Health Organization (WHO) (1978) defines normal blood pressure as systolic readings <140 mmHg and a diastolic reading of <90 mmHg. They also define hypertension as a systolic blood pressure >160 mmHg and diastolic >95 mmHg. This leaves a grey area in the middle which the Framingham study calls borderline as mentioned above, but this borderline area is not free of risks.

A very recent report by the U. S. Department of Health and Human Services (1984) classifies an individual as having high normal pressure when their diastolic blood pressure is 85—89 mmHg. They recommend re-checking and monitoring their pressure within one year but definitive treatment is not required. They also define blood pressures >140/90 mmHg as hypertensive and recommend treatment for these people regardless of age.

Cumming et al. (1975) defined hypertension as a systolic blood pressure >145 mmHg and diastolic >90 mmHg. He and others (Cumming et al., 1975; Hellerstein and Franklin, 1984) also define a systolic blood pressure >220 mmHg during exercise as hypertension.

Kozlowski and Ellestad (1984) noted abnormal blood pressure responses to excercise. Failure of the systolic pressure to rise above 130 mmHg or fall by more than 10 mmHg during exercise is classified as abnormal. Also, if the diastolic pressure rises by more than 15 mmHg the blood pressure response was also called abnormal.

Labile hypertension (i.e. blood pressure elevated on some occassions and normal on others) has long been regarded as innoccuous when compared to fixed hypertension (i.e. constantly elevated)(Kannel et al., 1980b; Kannel et al., 1984). Recent work has shown labile hypertension to be no less important than fixed hypertension. Patients whose pressures are more labile have increased risk for CHD similar to those with less variable blood pressure. There is no question that persistently elevated pressure is associated with higher risk, however it is not safe to say that people with labile pressure are free from risk.

The Pooling Project (1978) clearly demonstrated the excess risk of elevated diastolic pressure. For men between the ages of 40 and 64 years the excess risk for elevated diastolic pressure as compared to a diastolic pressure of <76 mmHg are as follows in Table 1:

Table 1: Diastolic Blood Pressure

Diastolic Blood Pressure	CHD Risk
>94	181/1000 men
88-94	77/1000 men
80-88	50/1000 men

Similar results were found in the U.S. railroad workers (Keys, 1970). The prevalence of CHD in men with diastolic pressure >90 mmHg was 5.8 times greater than those with pressures <80 mmHg.

Isolated systolic hypertension is substantially less common than diastolic hypertension especially under the age of 45 (Kannel et al., 1980a). However, persons with isolated systolic hypertension have a substantially increased risk for CHD. Bruce et al. (1980) reports increased risk with systolic pressures >140 mmHg.

Hypertension demonstrates familial characteristics (Paffenbarger et al., 1983). College alumni were at an 83% greater risk of developing hypertension if they had one parent who was hypertensive when compared to those without parental hypertension.

Body weight is also related to hypertension (Irving et al., 1977; Paffenbarger et al., 1983). Hypertensive patients had significantly more body weight than normotensives. Men who were 20% or more over their ideal weight for height were at 78% greater risk of developing hypertension than lighter men. Paffenbarger et al. (1983) went on to show that it is obesity (ie. excess body fat), rather than weight per se that increases the risk.

Other risk factors such as hypercholesterolemia and smoking, have an additive affect with hypertension (Kannel, 1975). Hypertension has been shown to accelerate and intensify atherogenesis significantly in cholesterol fed animals (Stamler, 1979).

Intervention trials to lower blood pressure reinforce the importance of blood pressure as a risk factor. The Veterans Adminstration study (1970) demonstrated such a dramatic improvement in cardiovascular disease morbidity, for severely hypertensive patients over

controls receiving placebos, that the trial had to be stopped because it was unethical not to treat these severely hypertensive patients. In severely hypertensive patients (>164 mmHg systolic or >104 mmHg diastolic) the affectiveness of treatment in preventing terminating organic complications was 73%. These researchers felt there was little doubt about the effectiveness of antihypertensive medication in the severely hypertensive. However, they were less convinced in the mildly hypertensive.

The Hypertension Detection and Follow-up Program Cooperative Group (1979) investigated the response of mildly hypertensive subjects to specific antihypertensive therapy. They demonstrated a decrease in 5 year mortality from all causes of 17% when compared to the normal treatment group and a decrease in CHD mortality of 15% over this same period.

Additional work has been done to substantiate these findings (MRFIT, 1982; Puska et al., 1983). Treatment of mild hypertension (diastolic pressures between 90—104 mmHg) resulted in 46% fewer deaths from myocardial infarction and 45% fewer deaths from cerebrovascular disease.

CIGARETTE SMOKING

There is believed to be a distinct relationship between cigarette smoking and arterial thrombotic disease (Levine, 1973). Nicotine in cigarettes causes platelet aggregation and liberation of free fatty acids, not to mention, it's transient effects of increased heart rate and vasoconstriction.

The World Health Organization (1975; 1979) classifies smoking as a major and modifiable risk factor for ischemic heart disease. Cigarette smoking acts both independantly and synergistically with other major risk factors such as hypertension and hypercholesterolemia to increase the risk of CHD. Cessation of smoking significantly reduces the mortality rate from CHD compared to those who continue to smoke.

U.S. railroad workers had a death rate from CHD that was 428% higher in smokers, who smoked more than 20 cigarettes per day, than in non-smokers (Keys, 1970). The American

Heart Association (1960) reports that the death rate from CHD is 50—150% higher in heavy smokers. This data suggests that heavy cigarette smoking contributes to, or accelerates the development of CHD.

The Framingham data (Dawber, 1980; Kannel, 1981) demonstrated twice the risks for CHD in smokers over non-smokers. In addition, the age of onset of CHD was younger, by 2.5 years on average, in male cigarette smokers. The affects of cigarette smoking were most pronounced in young males especially under the age of 40. The risks of having a myocardial infarction, when smokers were compared to non-smokers, was 3 times greater for ages 30—39, 2 times greater for ages 40—49 and there was no difference for ages 50—59.

There appears to be a dose response relationship between the number of cigarettes smoked and the development of CHD (Kannel, 1981). The Pooling Project (1978) also demonstrated this phenomenon. A pack-a-day smoker has 2.6—4.0 times greater risk of a first coronary event than a non-smoker regardless of age. The risk rises rapidly above 1/2 pack /day, but 1/2 pack/day smokers still are at greater risk than non-smokers.

Angiographic studies also point out the relationship between CHD and cigarette smoking (Cramer et al., 1966). A higher rate of severe arterial damage in the walls of the coronary arteries occurs in smokers regardless of their serum lipids.

Cessation of smoking reduces ones risk of mortality from CHD (WHO, 1975). The risk decreases gradually with time, but it may take an estimated 5, 10, or even 20 years before the risk is equal to that of a non-smoker (Kannel, 1981; Kannel et al., 1984). The greatest decrease in risk, up to 50%, occurs within the first year after stopping the cigarette habit (Kannel, 1981). It is estimated that a decline in cigarette smoking in the general population could reduce mortality from CHD by 14—42% depending upon the assumptions made about dose response curves to CHD (Kannel, 1981).

Coronary heart disease rates are down 24% from 1968 to 1976 and the number of cigarette smokers has decreased since 1964 by 25% in males and 11% in females (Stamler, 1979). Smoking intervention programmes are generally unsuccessful, but the MRFIT group (1976)

demonstrated a decrease over six years in the number of smokers from 59 to 46%. Therefore, modification of this lifestyle factor is possible, and it does make a noticeable difference.

EXERCISE

Physical activity during working hours and/or leisure time has been linked to the prevention of CHD. Morris et al. (1953a) were pioneers in this area of research with their work on the London busmen and conductors. They found a decreased incidence of CHD in the conductors when compared to the bus drivers. They proposed that this was due to the added physical demands of the conductor's job, and that their physical activity was having a protective effect against CHD. They went on to study letter carriers against inside postal workers and the same phenomenon occured (Morris et al., 1953b). The letter carriers had a lower incidence of CHD; and when it did occur it appeared later in life. However, no attempt was made to control for confounding variables such as job stress or self-selection into a particular job due to underlying disease.

In later work by Morris et al. (1966 ; 1973), some of these confounding variables were analyzed. It was found that drivers had higher systolic blood pressure and higher serum lipids than the conductors. However, in the final analysis they still concluded that physical activity does play a role in promoting cardiovascular health.

Paffenbarger and Hale (1975) studied the physical activity level during the working hours of San Francisco longshoremen. Attempts were made to control for self-selection into easier jobs due to underlying desease. In spite of this, the highly physically active group developed significantly less heart disease than the moderately or lightly active groups (26.9/10,000, 46.3/10,000, 49.0/10,000 respectively). The crucial level of physical activity that was required during working hours was 5.2 kcal/min of energy. This risk protection was found to be independant of smoking history, systolic blood pressure, obesity and diabetes mellitus.

Autopsy studies also support the premise that physical activity is protective against CHD. These studies showed more healed infarcts, fibrous patches, scars and coronary

occlusions in individuals who engaged in light as opposed to heavy job related physical activity (Fox et al., 1971).

Leisure time physical activity has also been associated with a lower incidence of CHD (Morris et al., 1973; Paffenbarger et al., 1978b). Expending less than 2,000 kcal/week in leisure physical activity had a 64% increased risk of CHD over those who utilized greater than 2,000 kcal/week (Paffenbarger et al., 1983). The affect of physical inactivity combined with other risk factors such as cigarette smoking, obesity and hypertension magnified the risk of developing CHD (Paffenbarger et al., 1978a).

Peters et al.(1983) carried out a follow-up study over a 10 year period on a group of men who were classified into fitness categories based upon a test of cardiovascular fitness. They found that the unfit men were twice as likely to have a heart attack as their fit counterparts; and when 2 or more risk factors were added to this lack of fitness the risk increased to 6 times.

The Framingham data (Dawber, 1980) also supports the case for physical activity. Sedentary individuals had approximately 3 times the rate of CHD when compared to the most active group. However, the benefit from moderate activity was less pronounced. It appears that an excess energy expenditure of 400—500 kcal/day is required to offer protection from CHD (Fox et al., 1971). The absolute minimum would be 1 hour of aerobic activity spread over 3 days in the week (Nora et al., 1978).

The multifactoral beneficial effects of exercise are well demonstrated in a report by Lopez et al. (1974). An intense programme of short duration resulted in significant decreases in serum triglycerides, LDL cholesterol and increases in HDL levels.

The benefits of exercise are not as pronounced when heart disease is already present (ie. post-infarction) (National Exercise and Heart Disease Project, 1981; Rechnitzer et al., 1983). The activity in these groups was not harmful but it failed to have any significant effect on mortality rates or re-infarction rates.

FAMILY HISTORY

Familial aggregation of CHD, particularly when it results in the early onset of the disease, is well recognized. Risk factors such as hypertension, hyperlipidemia, obesity, smoking and impaired glucose tolerance tend to cluster in families (Robertson, 1981; Kannel et al., 1984). Persons who develop CHD are concentrated in fewer families than chance would dictate. A positive family history of a first degree relative with CHD increases the risk of developing CHD by 2—5 times over someone with a negative history. Nora et al. (1980) found that the highest risk ratio for CHD was in persons with a family history of a first degree relative who had the onset of CHD before age 55, and then secondly a first degree relative with disease before age 65. Also associated with increased risk of CHD was a family history of stroke and hypertension. Even after eliminating familial hyperlipidemia the inevitability of CHD in persons with a positive family history before age 55 was 56%. Familial hyperlipidemics are not common but they occur more frequently among patients with CHD. In this study 15% of patients age 35—54 who survived a myocardial infarction had a familial lipid disorder.

The early work by Morris et al. (1966) discovered this familial trend of CHD. Morris et al. (1966) found that a positive family history resulted in a CHD incidence rate of 9.1/1000, whereas a negative history had an incidence rate of 5.9/1000 for all ages.

A Finnish study (Rissanen and Nikkila, 1977) further demonstrated the inevitability of CHD. The risk of dying from CHD before age 65 was 5 times greater for fathers of cardiac patients than for fathers of controls. For brothers of cardiac patients the risk of developing fatal or non-fatal CHD before age 65 was 5 times greater than for sisters of controls. The clustering affect of CHD in families was also seen in this study. In 25% of the families of cardiac patients there were 3 or more cases of CHD before age 65. Approximately 50% of angina patients had at least one sibling who manifested CHD.

Risk factors, for example hypertension and hyperlipidemias, also tend to show a familial predispostion (Deutscher et al., 1966; Rissanen and Nikkila, 1977; Paffenbarger et al., 1983). The data suggests that genetic factors play an important role over and above the

environment. It is possible to identify an individual who is at risk on the basis of family history of risk factors, and manifest CHD (Deutscher et al., 1966). Once such a person is identified lifestyle modification of detrimental risk factors is possible and recommended.

DIABETES MELLITUS

Cardiovascular disease causes death in 57% of diabetics and only 32% of non-diabetics (Ducimetiere et al., 1979). Coronary atherosclerosis is more severe in all ages and occurs at a younger age in insulin-dependent diabetics, but coronary heart disease is not inevitable in all diabetics and not all coronary arteries become involved. There is however, something unique about diabetes that increases the risk of CHD over and above other risk factors especially in women (Stout, 1981).

According to the Framingham study (Kannel and Mc Gee, 1979; Dawber, 1980; Kannel et al., 1984) the risk of cardiovascular mortality, including CHD, doubles in diabetics. Women seem to be at greater risk than men, which is the opposite to the other risk factors. The incidence of CHD in diabetic men is 2 times and in women 3 times greater than their non-diabetic counterparts. The risk of CHD decreases with advancing age but it is never the same as a non-diabetic (Stamler et al., 1979).

The risk is magnified by other co-existing risk factors, but it does not appear that diabetics cope less well with risk factors than do non-diabetics (Kannel and Mc Gee, 1979). There does not appear to be any statistical interaction between diabetes, smoking, hypertension, serum cholesterol and obesity.

Glucose intolerance rather than overt diabetes mellitus has also been investigated and there appears to be a positive non-linear relationship between blood sugar and mortality rates for CHD (Fuller et al., 1979; Pyorala et al., 1979; Ducimetiere et al., 1979). Mortality rates are significantly higher in the top quintiles. Specifically, individuals with a 2 hour post-prandial blood sugar above the 98th percentile for their age, have twice the risk of developing CHD over those below this level. There appears to be a threshold above which the risk increases and below

which there is no effect on mortality rates (Fuller et al., 1979; Ducimetiere et al., 1979).

The 2 hour post-prandial blood sugars required to put an individual into the 98th percentile are not exceptionally high. For males age 40—64 the critical value is 110 mg/dl (6.1 mmol/l) (Fuller et al., 1979). It is also important to note that the relationship between blood sugar and CHD is with 2 hour post-prandial sugars and not fasting sugars. Fasting blood sugars did not show any significant relationship to CHD (Pyorala et al., 1979).

Despite the statistics indicating the increased risk in diabetics, multivariate analysis failed to show blood glucose as an independant risk factor for CHD in non-diabetics (Pyorala et al., 1979; Fuller et al., 1979; Ducimetiere et al., 1979). Stenhouse et al. (1979) and Stamler et al. (1979) both concluded that asymptomatic hyperglycemia does not act as a significant risk factor for increased mortality from CHD.

Several ideas have been put forth to explain the relationship between diabetes mellitus and CHD. Increased triglycerides and increased insulin levels are two of the most commonly sited (Fuller et al., 1979; Ducimetiere et al., 1980). High triglyceride levels often accompany elevated serum glucose levels. Also high fasting insulin levels have been shown to be positively associated with CHD. It is suggested that the excess insulin, which enhances cholesterol synthesis for a given level of glucose, is the key variable for predicting CHD complications.

OBESITY

Insurance company statistics suggest that excess body weight is associated with an increase in general mortality (Kaplan and Stamler, 1983). They also suggest that men and women who reduce their weight to normal and maintain their new weight reduce their risk to within normal limits for their age and sex.

U.S. railroad workers demonstrated an increased incidence of CHD in persons with elevated body weight and sum of skinfolds (Keys, 1970). The Framingham data (Dawber, 1980) supports their claim. Males who were 20% or more above the median weight for age had twice the risk of developing CHD over those below the median. Similar findings were reported

for females. The incidence of CHD was 50% in all obese persons regardless of age and sex. In addition, body weight showed a strong positive relationship to both systolic and diastolic blood pressure.

The Pooling Project (1978) found an increased risk for men only in the age groups 40—44. Here the relative risk was 2.1. The remaining age groups did not demonstrate any increased risk of CHD with increased body weight.

The body weight is closely allied with other risk factors. Paffenbarger et al. (1983) noted that it is obesity (ie. body fat) that is the important factor not just excess body weight to height. Using the body mass index (BMI) as a measure of obesity, they found that a BMI of 32+ (greater than 30 is obese) was associated with a 24% increased risk of hypertension.

Kannel et al. (1984) reported that obesity is strongly linked to hypertension, hyperinsulinemia, impaired glucose tolerance, decreased HDL, hypertriglyceridemia, and hyperuricemia; all of which have been associated with CHD. Kaplan and Stamler (1983) also commented that obesity is linked with low HDL levels and that weight reduction and physical activity are associated with higher HDL levels. They emphasized that physical activity should be part of any weight control programme.

Others have noted similar relationships. For example, obese persons have elevated triglyceride levels and decreased HDL levels (Gordon et al., 1977; AHA,1984). Also body mass index is positively related to blood glucose levels (Pyorala et al., 1979). Finally, hypertensive patients have significantly more body weight than normotensives (Irving et al., 1977).

Obesity is very stongly interrelated to other risk factors and it is often difficult to separate the effect of obesity alone. Some report that obesity is in fact not an independant risk factor (Cramer et al., 1966; Kaplan and Stamler, 1983). The synergistic effect of body weight with other risk factors is demonstrated below in Table 2:

Table 2: Body Weight as a Risk Factor (Stamler, 1979)

Risk Factor	Mortality Ratio
overweight alone	1.4
overweight + SBP† >130	2.3
SBP† >130 + smoking	2.1

† SBP refers to Systolic Blood Pressure

ALCOHOL

The issue of alcohol and CHD is somewhat confusing at present. Several authors report an inverse relationship between alcohol and CHD (Klatskey et al., 1974; Hennekens et al., 1978 and 1979; Kannel et al., 1984). However, others report a lack of association between CHD and alcohol consumption (Wilson et al., 1980).

Klatsky et al. (1974) noted a larger number of non-drinkers than drinkers in persons suffering from their first myocardial infarction. There was an inverse relationship between alcohol consumption and incidence of myocardial infarction no matter what amount of alcohol was consumed. Interestingly, they also found a greater number of heavy drinkers (6 times) in the smokers than non-smokers. They suggest that there may be confounding variables in the alcohol relationship to CHD, such as personality trait and ethnic background.

Hennekens et al. (1978 and 1979) found that the preventative effect was restricted to moderate to light (< 2 oz/day) alcohol consumption. They did not find any association between CHD and heavy alcohol consumption. The risk ratio of light consumption versus no consumption was 0.4 (range 0.3 to 0.6). Again, they suggest that confounding variables such as personality type (ie. type A or B) may have an effect on these results.

The Framingham data (Stason et al., 1976; Kannel et al., 1984) supports the inverse relationship between alcohol consumption and non-fatal CHD. Again, this negative correlation appears stronger in smokers than non-smokers with relative risk ratios of 0.6 to 1.3

respectively. (Stason et al., 1976). The importance of this finding has yet to be explained.

The suggestion has been made that the protective effect is due to the increase in HDL levels and the decrease in LDL levels caused by alcohol (Castelli et al., 1977). As discussed previously elevated HDL levels protect against CHD.

The major difficulty with assessing alcohol consumption and CHD is the inaccuracy of reporting by the subjects or their relatives (Stason et al. 1976; Wilson et al., 1980). People are reluctant to admit to their actual alcohol consumption for fear of being labelled an alcoholic.

MULTIPLE RISK

Coronary heart disease is mulifactorial and the integration of these risk factors is required to get a clear picture of one individual's risk. A composite risk score can be derived for an individual by using a multiple regression equation that was developed from the Framingham data. This equation was developed through multiple regression analysis and probability statistics of numerous CHD risk factors (Kannel et al., 1976). The American Heart Association (1973) has devised an easy method, based on the Framingham data, for estimating an individual's risk by incorporating such risk factors as smoking, blood pressure, serum cholesterol, age, sex and glucose intolerance. This method will be applied to the data in the current investigation to estimate each subject's combined risk for CHD.

By way of example, consider the case of a normotensive, normoglycemic 45 year old male. If he was a non-smoker with a serum cholesterol of 185 mg% his risk of developing CHD in the next 6 years would be 1.8/100; and if this same man smoked his risk would go up to 2.7/100. Now if he was a non-smoker with a serum cholesterol of 285 mg% then his risk would be 4.6/100; and if he was a smoker with a serum cholesterol of 285 mg% then his risk would be 7.0/100 of developing CHD in the next 6 years. Finally, if this 45 year old male smoker with elevated serum cholesterol also was hypertensive his risk would increase to 11.7/100. The risk increases by 1.5, 2.6, 3.9 and 6.5 times respectively over the various risk factor combinations. This example clearly illustrates how the risk factors combine to increase one's risk of

developing coronary heart disease.

SUMMARY

In summary risk factor analysis is a multifactorial problem and it is often difficult if not impossible to isolate the amount of risk associated with each factor. However, each of these risk factors has some identifiable association to CHD and many of these risk factors are easily modified through lifestyle changes; one of which is increased physical activity. Therefore, these risk factors will be examined within the context of their relationship to one's level of fitness as determined in a fitness appraisal centre.

Chapter III

METHODOLOGY

SUBJECTS

The subjects for this study were male and female adults, age 17—72, from the general population of the Edmonton area, who voluntarily presented themselves for a Fitness and Life Style Appraisal at the University of Alberta Fitness Unit.

FITNESS UNIT PROCEDURES

All subjects completed a consent form and the PAR-Q exercise readiness questionnaire (Chisholm, 1978) prior to commencing the test. Each subject was required to be PAR-Q negative prior to proceeding with the fitness appraisal.

The general test of fitness included measurements of height, weight, skinfolds for prediction of body composition using the Harpenden calipers, grip strength, trunk flexion, sit-ups, push-ups and a measurement of cardiovascular fitness. Finally, each subject completed the health hazard appraisal questionnaire (Health and Welfare Canada, 1980).

The percent body fat calculations were based on the sum of 4 skinfolds from the bicep, tricep, suprailiac and subscapular sites. The sum of skin folds was then applied to the Durnin formula to predict the percent body fat (Durnin and Wormersley, 1974).

The cardiovascular test of fitness was an eight minute bicycle ergometer test (Astrand and Rhyming, 1954). The maximal oxygen consumption (VO_2MAX) for each subject was predicted from the heart rate response to a specific workload using the Astrand nomogram (Astrand and Rhyming, 1954). The VO_2MAX obtained from the nomogram was corrected for age (Astrand, 1960) and then each subject's VO_2MAX was assigned a percentile using the Swedish norms (Astrand, 1960). The subjects were classified into three groups according to Table 3.

Table 3: Cardiovascular Fitness Criteria

Percentile	Fitness Category
<5—30	Low
35—65	Moderate
70—>95	High

HOSPITAL PROCEDURES

The low and high fitness groups only, were referred to the Cardiac Rehabilitation Unit at the University of Alberta Hospital for further assessment. The procedure at the hospital involved a detailed medical history, including family history of CHD, stroke, hypertension and diabetes mellitus, alcohol and cigarette consumption, and physical activity, followed by a physical examination by a physician. Following this an exercise stress test on the treadmill to 90% of their age predicted maximum was carried out. The Bruce protocol (Bruce, 1971), and a Marquette Case computer assisted ECG analyzer was used to analyze the results. The blood pressure was recorded using a mercury manometer at rest, during the last minute of each work load during exercise, immediate post-exercise and in the fourth minute of recovery. Blood pressure was taken using a mercury manometer with systolic pressure being read at the first Korotkoff sound and diastolic at the fourth Korotkoff sound (ie. muffling of the sound). Blood pressure was categorized as hypertensive by the criteria presented in Table 4.

Table 4: Hypertensive Blood Pressure Criteria

State	Characteristic
Rest	SBP† >140 DBP‡ >90
Exercise	SBP† >220

† SBP refers to Systolic Blood Pressure
‡ DBP refers to Diastolic Blood Pressure

The blood pressure response to exercise was also load related;so that, a systolic blood pressure of 220 mmHg on the first workload is a hypertensive response, but it may not be a hypertensive response if it occurs on the fifth workload. Individuals were also classified as having abnormal blood pressure response to exercise when the systolic blood pressure failed to rise above 130 mmHg or fell by more than 10 mmHg; or when the diastolic pressure rose by 15 mmHg or more.

Each subject was asked to return after a 12 hour fast for a resting, standardized electrocardiogram (ECG) and a blood sample in which serum lipids and glucose were measured. The serum lipids and glucose were analyzed in the laboratory at the University of Alberta Hospital. Serum glucose was measured by the American Monitor Parallel methododology (Trinder,1969). The triglycerides were analyzed by the BMC triglyceride enzymatic method (Bergmeyer,1974) and the cholesterol by the BMC cholesterol-C system CHOD-PAP method (Siedel,1981). The HDL was measured by the NIH 75-628 publication lipid research program method (1974). The lipid ratio utilized was the LDL + VLDL/HDL ratio.

Table 5 describes the limits for the various blood parameters. A person with blood parameters falling outside of these limits was classified as having abnormal (ie. increased or decreased) lipids or fasting glucose values.

Table 5: Blood Lipid and Glucose Limits

Serum Cholesterol	> 6.5 mmol/l (250 mg%)
Serum Triglycerides	> 2.0 mmol/l (77 mg%)
Serum HDL	< 0.9 mmol/l (34 mg%)
LDL + VLDL/HDL Ratio	> 4.0
Fasting Serum Glucose	> 6.0 mmol/l (108 mg%)

The criteria for determining obesity was based on the Canadian norms from the Standardized Test of Fitness (Fitness Canada, 1981). The above average category for age and sex was used to determine the cut-off point for obesity. Subjects were classified as obese if their percent body fat was above this cut-off point. Table 6 outlines the body fat classifications.

Table 6: Obesity Criteria

Age	Sex	% Body Fat
15-29	M	>19.9
	F	> 27.9
30-39	M	>23.9
	F	> 31.9
40-49	M	>27.9
	F	> 35.9
50-69	M	>29.9
	F	> 37.9

Each subject was questioned about their leisure time activity level while undergoing the treadmill test at the hospital. The activity levels were categorized according to the individual's

daily aerobic activity. Table 7 demonstrates this classification system.

Table 7: Classification of Physical Activity

Level	Activity
1	Sedentary
2	Aerobic Activity <3 Times Per Week
3	Aerobic Activity 3 Times Per Week
4	Aerobic Activity >3 Times Per Week

The alcohol consumption was quantified into the number of drinks per week regardless of the type of alcohol consumed. The subjects' alcohol consumption, reported at the fitness unit on the Health Hazard Appraisal questionnaire and at the hospital during the physician interview, was recorded.

The subject's cigarette smoking habit was catergorized into smoker, non-smoker, or ex-smoker. In order to qualify as an ex-smoker the subject must have abstained from smoking for at least one year.

The family history was deemed to be positive when a first degree relative (ie. parent or sibling) suffered from coronary heart disease, diabetes mellitus, hypertension or a stroke. The age of onset for these diseases was not taken into consideration.

STATISTICAL ANALYSIS

The Chi-square test for independence was performed on the data points where the subjects were categorized by possesion or absence of a particular risk factor. Oneway analysis of variance between levels of fitness was executed on the means of the following parameters: blood lipids and glucose values, blood pressure values, age, height, weight, percent body fat,

alcohol intake and VO_2MAX. In addition, a T-test on the previously mentioned means was obtained between the sexes. A Pearson product-moment correlation between VO_2MAX, alcohol intake and fitness classification at the hospital and the fitness unit was executed.

The probability (per 100) of developing CHD in 6 years was estimated for each subject using the American Heart Association Coronary Risk Handbook (1973). All subjects were presumed to be left ventricular hypertrophy negative by ECG criteria, because this was outside the limits of the current study. The means and standard deviations for these probabilities were calculated and a T-test was applied to these means with respect to fitness level and sex.

Chapter IV

RESULTS AND DISCUSSION

As demonstrated in Table 8, out of the 260 subjects originally tested 75% of the clients (N=194) at the fitness unit accepted further testing at the hospital. Of the 173 subjects who were actually tested at the University of Alberta Hospital stress testing lab, 42% were classified in the low fitness, and 58% were classified in the high fitness group.

Table 8 : Subjects Tested vs. Fitness Classification

	Low Fitness (<5—30%ile)	High Fitness (70—95%ile)	Total
Subjects Tested at the Fitness Unit	108 (42)	152 (58)	260 (100)
Subjects Referred for Stress Testing	82 (32)	112 (43)	194 (75)
Subjects Refusing Stress Testing	26 (10)	40 (15)	66 (25)

( ) percentage of total number tested.

An additional 21 subjects who volunteered for further testing at the hospital were lost to follow-up for various reasons. The most common reason was an inability to arrange appointment times suitable to the client and the stress testing lab. Table 8 lists the number of subjects that were tested at the fitness unit and the stress test lab.

AGE AND SEX

Table 9 gives an additional breakdown of these 173 subjects with respect to fitness level and sex. Proportionately more subjects fell into the high fitness category than the low fitness category, and this was especially true of the female population, (76%) compared to males (48%). This discrepancy may be due to the small number of females tested and thus interpretation of the data may be confounded. If there were equal proportions of men and women in the referral and refusal categories and equal numbers in the omitted middle fitness category, there was a distinct absence of women in the sample. The female sample in this investigation was definitely skewed towards the high fitness category. Due to the small number of females in the low fitness group interpretation of their data must be done so with extra caution.

Table 9 : Percentage of Subjects Tested at U of A Stress Testing Lab with their Mean Ages and Standard Deviations

	Both Sexes	**Males**	**Females**
Low Fitness (<5—30%ile)	42.0±10.5[1] (44)	41.8±10.8[1] (52)	42.4±9.1[1] (24)
High Fitness (70—95%ile)	32.4±9.2 (56)	33.1±9.0 (48)	31.4±9.5 (76)
Total Sample	36.6±10.8 (100)	37.6±10.8 (70)	34.5±10.5 (30)

( ) percentage of subjects

[1] $p<0.01$ low to high fitness comparison

In addition, according to the Canada Fitness Survey 56% of Canadians are physically active and the numbers of men and women are equal (Fitness and Amateur Sport,1983). Since the present investigation does not deal specifically with activity but rather the number of Canadians who voluntarily seek information about their level of fitness, this may be the reason for the differences noted between the female and male results. Perhaps there is a difference in health consciousness between the sexes. It may be that men are either more concerned about their fitness or that women are staying away from these testing centers for other reasons. Gruneau et al. (1976) and Hall and Richardson (1982) found that women participate in sport less than men for numerous reasons. If one extrapolates from sport to exercise and fitness in general, their findings may apply to the current study. They found that women do not have any less interest than men, but other things in their lives have a higher priority, such as family and careers. Women tend to put their own interests and pursuits behind their commitments to their families. Unless women are well supported through family, peer groups, social groups or husbands they succumb to family and job pressures. More women than men expressed a dissatisfaction about their level of activity but they were less likely to do anything about it. Perhaps these same feelings can be extrapolated to the fitness testing environment.

With regard to age, the low fitness group was significantly older than the high fitness group (Table 9). In addition to the data presented in Table 9, it should be noted that over 60% of the subjects were under the age of 40 and 30% were under the age of 30. This relationship held true when the population sample was divided into the sexes. This relationship may be supported by data from the Canada Fitness Survey, since younger Canadians tend to be more active than the older groups (Fitness and Amateur Sport #5,1983). This would indeed influence the fitness levels of a population. The age results confirm the work of Gibbons et al. (1983) who found that the unfit women in their study were significantly older than the fit women.

The subjects in this investigation were generally very young and yet significant risk factor abnormalities were detected in these young people. Since these abnormalities presented at

a young age, correction of these problems may help to lower their risk of CHD. Counselling of these young people about a healthier lifestyle and risk factor modification may lead to decreased morbidity and mortality from CHD in their later years.

SERUM LIPIDS

More males (n=21) than females (n=2) had elevated triglycerides, regardless of fitness classification. The remainder of the lipid parameters did not show any differences in terms of the proportion of subjects with lipid values outside the established criteria. There was a distinct absence of persons with abnormally low HDL values despite several with elevated ratios. This was most likely due to the strict criteria used to establish the lower limit of normal for the HDL fraction.

The low fit classification showed higher ($p<.01$) values for triglycerides, cholesterol, lipid ratio and fasting blood sugar plus lower HDL values (Table 10). This phenomenon persisted for males but not females when the fitness classifications were sub-divided by sex. Low fit males had higher ($p<.01$) values, for triglycerides, cholesterol and lipid ratio plus lower HDL values ($p<.05$). Lower fit females demonstrated higher values for serum triglycerides ($p<.01$) with minimal changes in the other lipid parameters ($p>.05$). When comparing males to females, regardless of fitness level, the males had higher triglyceride, cholesterol ($p<.01$) and ratios plus lower HDL values ($p<.01$;Table 10). Whether these differences reflect the small sample size for the female population or true differences is uncertain; however women are less prone to CHD and this may be due to less severe problems with serum lipid aberrations. The latter suggestion is warranted in light of the HDL levels.

The female mean HDL level was 1.04 mmol/l compared to 1.40 mmol/l for the males. This difference of 0.36 mmol/l (13.85 mg%) is in close agreement with Castelli et al. (1977) who found HDL levels to be 10 mg% (0.26 mmol/l) higher on average, in females than males. Castelli et al. (1977) also found a difference of 3—4 mg% (0.8—1.0 mmol/l) between persons with CHD and persons without CHD. The current investigation demonstrated a difference in

Table 10: Means and Standard Deviations for Blood Parameters

						Males		Females	
	Total Sample	Males	Females	Low Fit	High Fit	Low Fit	High Fit	Low Fit	High Fit
Triglycerides (mmol/l)	1.34 ±0.93	1.49[1] ±1.05	1.00 ±0.47	1.74[2] ±1.20	1.02 ±0.48	1.83[3] ±1.28	1.12 ±0.55	1.38[4] ±0.65	0.86 ±0.30
Cholesterol (mmol/l)	4.90 ±1.05	4.96 ±1.05	4.76 ±1.05	5.25[2] ±0.95	4.61 ±1.05	5.29[3] ±0.99	4.58 ±0.98	5.05 ±0.73	4.66 ±1.16
Lipid Ratio (mmol/l)	2.44 ±1.00	2.65[1] ±0.99	1.97 ±0.86	2.92[2] ±0.93	2.04 ±0.87	3.08[3] ±0.94	2.16 ±0.79	2.26 ±0.51	1.86 ±1.00
HDL (mmol/l)	1.48 ±0.35	1.40[1] ±0.31	1.04 ±0.37	1.38[2] ±0.30	1.56 ±0.37	1.33[5] ±0.28	1.47 ±0.33	1.58 ±0.31	1.70 ±0.40
Fasting Blood Sugar (mmol/l)	4.95 ±0.49	5.02[1] ±0.49	3.90 ±0.47	5.05[2] ±0.56	4.87 ±0.42	5.09 ±0.59	5.00 ±0.35	4.90 ±0.42	4.74 ±0.49

[1] $p<0.01$ male to female comparison
[2] $p<0.01$ low to high fitness comparison
[3] $p<0.01$ males, low to high fitness comparison
[4] $p<0.01$ females, low to high fitness comparison
[5] $p<0.05$ males, low to high fitness comparison

mean HDL values between the low and high fit subjects of >1.0 mmol/l in every case.

These relationships are consistent with previous work in the field on males alone (Hickey et al., 1975; Cooper et al., 1976; Erikssen et al., 1981). Gibbons et al. (1983) also demonstrated a significantly higher triglyceride value in the unfit women. They also found significantly lower HDL values and higher ratios in the unfit women. The current investigation failed to show significance in these latter fractions, however there is agreement on the lack of a significant relationship between serum cholesterol and fitness in women.

Hickey et al. (1975) and Cooper et al. (1976) demonstrated significantly lower serum cholesterol levels in the fit person, as compared to the less fit, in their studies. They also found this relationship to be independent of age. Erikssen et al. (1981) did not find any difference in

the serum cholesterol values, but they were the only group to study the lipid fractions and they did find higher HDL levels in their fit subjects. Shephard et al. (1980) also failed to demonstrate any lowering of the serum cholesterol values in their employee fitness participants. They speculated that it requires more intense exercise than what was undertaken by these groups. The subjects were involved in 30 minute sessions 3 times a week; but only 15 minutes of each session could be classified as intense aerobic work.

Both Cooper et al. (1976) and Erikssen et al. (1981) noted significantly lower serum triglycerides in the high fit groups and these results were independent of age. Attempts to study the lipid fractions and ratio were not carried out by these authors; but since the ratio involves the cholesterol, tryglyceride and HDL components, it naturally follows that significant differences between ratios in the low fit compared to high fit groups would occur when the individual components show significant differences.

The question now arises: what does one do with this information in the fitness environment? It is important to consider the value of this information in terms of counselling the subjects after their fitness test. All clients who present themselves to the fitness unit are presumed healthy and are PAR-Q negative, yet abnormalities in lipids which play a major role in the development of CHD are present. Most people have no idea about these abnormalities until a calamity happens such as a myocardial infarction. Even though blood samples are not taken during a routine fitness test, persons classified as unfit are at a greater risk of having these problems; so effective counselling about exercise, diet and lifestyle modifications becomes even more important. The mean ages of both low fit and high fit groups could be considered young even if the low fit group was significantly older. This also means that there were considerable numbers of people even younger than the mean ages who may have these abnormalities. Perhaps by encouraging lifestyle changes to clients in their twenties and thirties a lowering of the incidence of CHD will occur for these people when they reach their forties and fifties. It also appears that this information is equally important for both sexes.

HYPERTENSION

When the entire population is considered, the low fit persons had higher resting systolic and diastolic pressures compared to the high fit persons ($p<.01$;Table 11). This relationship persisted in the female subjects but not for the males. There were minimal differences in the mean exercise systolic blood pressure in any of the groups ($p>.05$; Table 11).

Table 11: Means and Standard Deviations for Blood Pressure

						Males		Females	
	Total Sample	Males	Females	Low Fit	High Fit	Low Fit	High Fit	Low Fit	High Fit
Resting SBP† (mm Hg)	121 ±15	124[1] ±14	113 ±14	125[2] ±13	118 ±15	125 ±13	123 ±15	125[3] ±13	110 ±12
Resting DBP‡ (mm Hg)	80 ±9	82[1] ±9	76 ±10	83[2] ±8	78 ±10	83 ±9	81 ±9	82[4] ±6	75 ±10
Exercise SBP† (mm Hg)	179 ±22	187[1] ±18	161 ±18	181 ±19	178 ±23	183 ±18	191 ±19	170[4] ±24	159 ±14

† SBP refers to Systolic Blood Pressure
‡ DBP refers to Diastolic Blood Pressure
[1] $p<0.01$ male to female comparison
[2] $p<0.01$ low to high fitness comparison
[3] $p<0.01$ females, low to high fitness comparison
[4] $p<0.05$ females, low to high fitness comparison

The results of this study agree with a previous study by Gibbons et al. (1983) which reported this relationship between fitness and blood pressure in women. They found significantly lower blood pressure in the fit women even though all of their subjects were normotensive. However, these results do not agree with Cooper et al. (1976) and Erikssen et al. (1981) who found significantly lower systolic and diastolic blood pressures in the fit males when compared to the unfit males. It would appear that physical fitness has a lowering effect on resting blood pressure even in normotensive adults.

Interestingly enough, the only group to show proporionately more subjects in either the

elevated blood pressure at rest or hypertension with exercise categories were the high fit males. This relationship is demonstrated in Table 12.

Table 12: Subjects with Elevated Blood Pressure

				Males				Females	
	Total Sample	Males	Females	Low Fit	High Fit	Low Fit	High Fit	Low Fit	High Fit
At Rest	18 (10)	16 (13)	2 (4)	10 (14)	8 (8)	9 (15)	7 (13)	1 (8)	1 (3)
During Exercise	7 (4)	6 (5)	1 (2)	1 (2)	6 (6)	0 (0)	6[1] (11)	1 (8)	0 (0)

( ) percentage of subjects
[1] $p<0.05$ males, low to high fitness comparison

Highly fit males had significantly more hypertensive responses to exercise than the unfit males. This may be anticipated; for men who exercise longer and reach a higher more difficult stage might be expected to respond with higher blood pressure. Also the current definition of hypertensive systolic blood pressure as >220 mm Hg maybe considered at the low end of the scale (ACSM guidelines,1980).

One would not expect major findings in the area of hypertension due to the criteria established by the fitness unit for testing normal subjects. All subjects must be PAR-Q negative which requires an absence of any history of hypertension. Secondly, any person with a resting blood pressure greater than 145/95 mm Hg would be disallowed from the testing procedure due to the restrictions imposed by the U of A fitness unit. This allows very little room for hypertensive subjects to meet the requirements for testing. The definition for hypertension in this study was 140/90 mm Hg; so there is very little difference between this value and the fitness unit cut-off criteria. Also anyone with labile hypertension could potentially have low pressure on the day of the fitness unit test and higher pressure during the

hospital test or vice versa.

Despite these methodological problems, the presence of higher resting values in the unfit population is consistent with the work of Hickey et al. (1975), Cooper et al. (1976) and Erikssen et al. (1981). Interestingly they tested only male subjects, and yet the current study did not show any significant differences between the males alone. The significant difference occurred in the general population and females alone.

The means for resting blood pressure are significantly different but they are still within the normal range, but they are very close to the new guidelines established by the U.S. Department of Health and Human Services (1984). However, if one considers the diastolic blood pressure at one standard deviation above the mean the low fit group then has a resting diastolic pressure above 90 mm Hg. Assuming a normal distribution, that would result in 16% of the unfit population being classified as hypertensive. A diastolic pressure greater than 90 mm Hg increases the risk of CHD by 5.8 times (Keys, 1970). The Pooling Project reported excess risk when the diastolic pressure rose above 76 mm Hg, and the higher the greater the risk. The Framingham data (Dawber, 1980) pointed out the danger of labile hypertension and borderline hypertension which are equally possible given the above data. Persons can easily vacilate between normal and slightly elevated blood pressure.

Again, these facts are important when counselling and advising clients about fitness and lifestyle especially in the case of the unfit. Mild changes in lifestyle to improve blood pressure may prevent the requirement for medication. Increasing activity plus dietary alterations of salt intake and calorie reduction may help to decrease obesity and hypertension.

CIGARETTE SMOKING

Table 13 demonstrates the proportions of smokers, non-smokers and former smokers in the female and male sub-groups. The proportions do not vary between the sexes or the fitness classifications ($p > .05$).

There appeared to be more non-smokers (including ex-smokers) in the high fitness

Table 13 : Smoking History by Sex

	Total Sample	Males	Females
Non-Smokers	107 (62)	73 (61)	34 (64)
Smokers	22 (13)	14 (12)	8 (15)
Ex-Smokers	44 (25)	33 (28)	11 (21)

( ) percentage of subjects
group regardless of sex (Table 14).

Table 14: Smoking History by Fitness Classification

			Males		Females	
	Low Fit	High Fit	Low Fit	High Fit	Low Fit	High Fit
Non-Smokers	41 (55)	62 (65)	33 (53)	38 (68)	8 (67)	24 (61)
Smokers	13 (18)	9 (10)	10 (16)	4 (7)	3 (25)	5 (13)
Ex-Smokers	20 (27)	4 (25)	19 (31)	14 (25)	1 (8)	10 (26)

( ) percentage of subjects

However, these differences in porportions were not statistically significant. The greatest proportion of subjects was in the non-smoking group regardless of fitness level, and the proportion of non-smokers was equal for both fitness classifications regardless of sex. There tended to be a greater proportion of smokers in the low fit groups for both sexes. However, the

females tended to have a greater proportion of the ex-smokers in the high fit group, whereas the males tended to have more ex-smokers in the low fit group. There were very few smokers in the high fit groups for either sex which indicates a healthy lifestyle of physical activity and avoidance of cigarette smoking.

The smoking trends in this study, despite the lack of statistical significance, are consistent with the work of Erikssen et al. (1981) and Gibbons et al. (1983) who found an inverse relationship between physical fitness and cigarette smoking in both men and women. Hickey et al. (1975) also found significantly fewer smokers in his group with heavy leisure time activity levels.

Only 13% of the sample population were smokers which is well below the reported national percentage of 31% (Fitness and Amateur Sport #21,1983). This gives an indication that our population was not representative of a normal Canadian population yet it may be representative of the Canadian population who seek advice about the level of fitness and are more health conscious. Perhaps as ones level of awareness toward personal fitness rises one begins to make changes by altering smoking habits.

EXERCISE

With regard to exercise habits, each exercise group was well represented but the greatest number of subjects appeared in the aerobic exercise 3 times a week group (Table 15 and 16). This was encouraging to see because it means that many people are making the effort to be physically active.

There was a significant difference in reported exercise levels between the 2 fitness groups, with the aerobically active individual being classified into the high fitness category. Yet as can be seen Table 16, several sedentary and low active people were classified in the high fit group and several very active people fell into the low fit group. This discrepancy probably resulted from 2 major factors:

1)the classification into the various fitness levels was found on questioning of the

Table 15 : Exercise Level by Sex

	Total Sample	Males	Females
Sedentary	42 (24)	31 (26)	11 (21)
< 3 times per week	33 (19)	21 (18)	12 (23)
3 times per week	69 (40)	45 (37)	24 (45)
>3 times per week	29 (17)	23 (19)	6 (11)

( ) percentage of subjects

Table 16: Exercise Level by Fitness Classification

			Males		Females	
	Low Fit	High Fit	Low Fit	High Fit	Low Fit	High Fit
Sedentary	31[1] (42)	8 (8)	26[2] (42)	3 (5)	5[3] (42)	5 (13)
< 3 Times Per Week	22[1] (30)	10 (11)	16[2] (26)	5 (9)	6[3] (50)	5 (13)
3 Times Per Week	19[1] (26)	50 (53)	19[2] (31)	26 (47)	0[3] (0)	24 (61)
>3 Times Per Week	2[1] (2)	27 (28)	1[2] (1)	22 (39)	1[3] (8)	5 (13)

( ) percentage of subjects
[1] $p<0.01$ low to high fit comparison
[2] $p<0.01$ males, low to high fit comparison
[3] $p<0.01$ females, low to high fit comparison

various subjects by the testers. This leaves room for error in interpretation of activity level by both the subject and the tester.

2)the classification into fitness levels may be incorrect. Submaximal testing is not one hundred percent accurate and this leaves room for error. This very topic will be discussed later in the paper.

Only 25% of the sample admitted to being totally sedentary and 56% reported an activity level consistent with aerobic fitness. This was consistent with the Canada Fitness Survey which reported that 56% of Canadians were physically active in their leisure time (Fitness and Amateur Sport,1983). The difficulty with the Canada Fitness Survey data is the definition of active; for it leaves considerable room for interpretation on the part of the subject. It would appear from this data that the message of improved health with fitness is getting across to many Canadians, however there is still lots of room for improvement.

The active persons tended to be younger than the non-active ones (Fitness and Amateur Sport #5,1983). The current data is consistent with this fact for the high fit group, even when subdivided into males and females, was significantly younger than the low fit group.

The Canada Fitness Survey also found that fewer active people were screened out of the fitness tests for medical reasons. Only 26% of the active people were screened out whereas 39% of sedentary people were eliminated (Fitness and Amateur Sport #8,1983). It appears that the active are more fit and healthier, but one cannot say for certain that activity causes good health, because perhaps the good health allows the increased activity.

FAMILY HISTORY

When comparing males and females, the proportions of persons with positive family histories were not significantly different (Table 17). However, when fitness classifications are considered, proportionately more people in the low fit category had positive family histories for coronary heart disease and diabetes mellitus ($p<.01$). This same relationship held true for the males alone when the fitness classifcations were subdivided into sex.

Table 17: Subjects with a Positive Family History

						Males		Females	
	Total Sample	Males	Females	Low Fit	High Fit	Low Fit	High Fit	Low Fit	High Fit
CHD	55	34	21	31[1]	23	25[2]	9	6	14
	(32)	(28)	(40)	(42)	(24)	(40)	(16)	(50)	(36)
Stroke	24	15	9	10	11	7	7	3	4
	(14)	(13)	(17)	(14)	(12)	(11)	(13)	(25)	(10)
Hypertension	59	39	20	22	35	19	19	3	16
	(34)	(33)	(38)	(30)	(37)	(31)	(34)	(25)	(41)
Diabetes Mellitus	29	21	18	18[1]	10	16[2]	5	2	5
	(17)	(18)	(15)	(24)	(11)	(26)	(9)	(17)	(13)

( ) percentage of subjects
[1] $p<0.01$ low to high fitness comparison
[2] $p<0.05$ males, low to high fitness comparison

It is well documented that a positive family history of CHD increases ones risk of developing CHD, in fact the risk increases by 2—5 times (Rissanen and Nikkila, 1977; Robertson, 1981; Kannel et al., 1984). This being the case it is even more important for persons with a strong family history to become physically active and not congregate in the low fitness category. This premise assumes that the benefits in mortality statistics illustrated by Morris et al. (1966), Paffenbarger and Hale (1975) and Dawber (1980) are in fact real. There is no evidence to suggest that physical activity will counteract the relationship of family history and heart disease, but there is also no evidence to the contrary.

The fact that diabetes mellitus also shows a relationship to low fitness levels is not totally surprising. Adult onset diabetes is related to a lifestyle of inactivity, obesity and overindulgence in large proportion of the population (Kaplan and Stamler,1983). Coronary heart disease also has this common lifestyle presentation in many cases. Children growing up in an environment, where this lifestyle represents their parental lifestyle, will be significantly

influenced by this environment (Braun and Linder, 1979). Environmental factors and influences in childhood play a large part in how one develops as an adult. These familial influences can paly a role in ones lifestyle as an adult and in many cases it takes a conscious decision to change these influences. This is not to downplay the genetic component in these diseases but lifestyle influences must also be considered. This is classic example of the nature/nurture issue.

DIABETES MELLITUS

Very few clients presented with elevated fasting blood sugars (Table 10). This is to be expected since the clients were PAR-Q negative prior to fitness unit testing. The few who did show abnormalities were only slightly above normal and could not be classified as having overt diabetes mellitus, but at best mild hyperglycemia. The values would have to be repeated with 2 hour post-prandial sugars before any accurate diagnosis could be made. However, the 3 subjects who did show abnormalities fell into the low fit, male classification.

The mean values for fasting blood sugar were different between the low fit and high fit populations as well as between males and females ($p<.05$). This information is of little value according to Fuller et al. (1979), Pyrorala et al. (1979) and Ducimetiere et al. (1979) who found a non-linear relationship between blood sugar and mortality rate for CHD. In order for blood sugar to be a factor one must be above the 98th percentile for their age. In addition, fasting sugars have shown little relationship to CHD. The main effects occur with post-prandial sugars.

OBESITY

Approximately 26% of the total sample was categorized as obese and this percentage was the same for both sexes. However, there were proportionately more obese people (46%) in the low fit group than the high fit group (10%) ($p<.01$). This difference became even more distinctive when the fitness groups were divided into the sexes. This phenomenon is well

demonstrated in Table 18.

Table 18: Anthropometric Data

						Males		Females	
	Total Sample	Males	Females	Low Fit	High Fit	Low Fit	High Fit	Low Fit	High Fit
Height (cm.)	173 ±9	178 ±6	164 ±8	175[1] ±8	172 ±10	177 ±6	178 ±6	166 ±10	163 ±8
Weight (kg.)	74 ±14	79 ±11	63 ±13	82[2] ±14	68 ±11	83[3] ±13	74 ±18	76[4] ±19	58 ±7
% Body Fat	24 ±7	22 ±6	29 ±6	27[2] ±7	22 ±7	26[3] ±5	18 ±5	35[4] ±7	27 ±5
Obese Subjects	46 (27)	32 (27)	14 (26)	34[2] (46)	9 (10)	27[3] (44)	3 (5)	7 (58)	6 (15)

( ) percentage of subjects
[1] $p<0.05$ low to high fitness comparison
[2] $p<0.01$ low to high fitness comparison
[3] $p<0.01$ males, low to high fitness comparison
[4] $p<0.01$ females, low to high fitness comparison

The classification system utilized was very liberal, so that one's percent body fat had to be well above average before one was classified as obese. The cut-off point was the 30th percentile or below for one's age and sex based on the Canadian norms for 1981. This result is not entirely unexpected since inactivity and obesity tend to go together (McDonough et al.,1970).

Table 18 also demonstrates the results of the anthropometric data. The mean heights, weights and percent body fat were different between the fitness classifications ($p<.01$). The fact that the height was significantly lower in the high fit group was due to a greater proportion of women in this group than in the low fit group. When the data is broken down by sex and fitness classification the low fit group is significantly heavier and fatter than the high fit group.

Obesity is linked to lifestyle diseases such as CHD, hypertension and diabetes mellitus (James,1977). Among the obese with no other medical impairment, it was found that the

number of deaths from cardiovascular disease was 50% more than those expected on the basis of normal mortality. In addition obese persons were 4 times more likely to be diabetic. More importantly a 20% reduction in weight in the obese can result in a 40% reduction in the chances of developing CHD. This is especially true for the young adult.

Previous work, by Hickey et al. (1975), Cooper et al. (1976) and Gibbons et al. (1983) supports the results demonstrated in this study. They all found significantly lower body weight and percent body fat in the fitter subjects regardless of sex.

The importance of this data is that improved cardiovascular fitness through regular exercise is one of the best ways to control one's body weight and body fat. According to the Canada Fitness Survey only 61% of adults (52% of men and 71% of women) say that weight control is important for personal health (Fitness and Amateur Sport #23,1984). Sixty-five percent of men and 54% of women who think weight control is important also feel regular exercise is important. Neither percentage is very high and the connection between weight control and exercise is not clear in the general public.

Too many fad diets are used to control weight when a moderate reduction in caloric intake plus moderate exercise will result in a far better and more permanent approach to weight control. A combination of exercise and dietary restrictions is a more effective approach to weight reduction than diet or exercise alone. In addition weight loss by diet alone causes significant loss of muscle mass, whereas, diet and exercise protects against loss of body fat (Katch and McArdle, 1983).

Gwimup (1975) found an average weight loss of 22 lbs. in obese women who exercised by walking for more than 30 minutes a day. This weight loss occurred over a one year period without any dietary restrictions on the part of the women on the study. In addition, the women commented that they did not like the exercise, but they did like the weight loss results and the feeling of well-being. They also did not feel weak or nervous; and they had greater strength and relaxation compared to dieting.

ALCOHOL

As can be seen in Table 19, males consumed on average more alcohol per day than females ($p<.01$). The actual amount consumed proved to be statistically significant only when one considers the fitness unit data; for, when one considers the hospital alcohol consumption statistics, the males did not consume significantly more alcohol than the females. The reverse situation occured when one looks at the alcohol intake with respect to fitness classification. Significantly more alcohol was consumed by the low fit people than the high fit people when one looks at the hospital alcohol reportings. This same difference appears in the fitness unit data but it is not statistically significant. The greatest problem with these results was the large variability in the responses to the question of alcohol consumption. The standard deviations were often larger than the means and this makes drawing reliable conclusions about alcohol intake difficult.

Table 19: Number of Drinks per Day

						Males		Females	
	Total Sample	Males	Females	Low Fit	High Fit	Low Fit	High Fit	Low Fit	High Fit
Reported at Hospital	3.7 ±4.1	4.7[1] ±4.6	1.9 ±2.0	4.3 ±4.7	3.2 ±3.5	4.7 ±4.9	4.1 ±4.1	2.3 ±2.9	1.8 ±1.7
Reported at Fitness Unit	4.1 ±5.1	4.7 ±5.5	2.9 ±4.0	5.1[2] ±5.8	3.1 ±4.0	5.2 ±5.9	4.0 ±4.7	4.7 ±5.8	1.9 ±2.0

[1] $p<0.01$ male to female comparison
[2] $p<0.05$ low to high fitness comparison

The value of alcohol consumption as a protective health influence when consumed in moderation is still up for debate. If it is protective, then the unfit should gain from this increased consumption but unfortunately the higher alcohol consumption fits into the pattern of obesity, inactivity and over indulgence.

Alcohol has been reported to increase HDL levels (Castelli et al.,1977). If the affect of cardiovascular disease protection is mediated through higher HDL levels one would expect higher levels in the unfit group, when in fact they were significantly lower. High alcohol consumption increases the daily caloric intake and can lead to obesity, which may be a contributing factor in the unfit.

As previously stated one of the major problems is to obtain an accurate estimate of one's alcohol intake. In this study a comparison between the reported alcohol intake at the fitness unit via a questionnaire, and to a physician by direct questionning was undertaken. The correlation between the 2 values was .73. One can conclude that only 50% percent of cases give a valid response to the question of alcohol consumption. This means that the reported alcohol consumption by an individual is variable and can make determining the benefits of moderate alcohol intake difficult to evaluate.

MULTIPLE RISK

Table 20 illustrates the results of the multiple risk factor analysis using the American Heart Association Coronary Risk Handbook (1973).

Table 20: Means and Standard Deviations for Probability (per 100) of Developing CHD in 6 Years.

			Males		Females	
	Low Fit	High Fit	Low Fit	High Fit	Low Fit	High Fit
Mean Risk (per 100)	2.61[1]	1.08	2.97[2]	1.28	1.05[3]	0.65
	±2.86	±1.37	±3.00	±1.64	±0.96	±0.32

[1] $p<0.01$ low to high fit comparison
[2] $p<0.01$ males, low to high fit comparison
[3] $p<0.01$ females, low to high fit comparison

The low fit subjects were at significantly greater risk of developing CHD within the next 6 years than the high fit subjects. This same trend persisted when the males and females were analyzed individually. The unfit persons were at 2.4 times greater risk of developing CHD in the next 6 years than the fit persons. Among the males the risk was 2.3 times greater for the unfit, and yet only 0.7 times greater for the unfit females over the fit. This data lends support to the previous evidence, that the physically unfit subjects are at significantly greater risk of developing CHD than the physically fit. The importance of this data is that it incorporates many of the risk factors into one estimate of an individual's risk.

SUMMARY OF RISK FACTOR DATA

In the previous sections the relationships between fitness and CHD risk factors were discussed. A common theme appeared throughout the discussion and that was one of lifestyle. Diseases such as coronary heart disease, diabetes mellitus and hypertension all have an element of unhealthy lifestyle to them. It was evident that the low fit group from the community based fitness appraisal centre expressed many of these undesirable qualities, such as higher serum cholesterol values, higher resting blood pressure, greater body weight and body fat, greater alcohol consumption and a greater likelihood of having a positive family history for coronary heart disease and diabetes mellitus.

An unhealthy lifestyle is known to be related to CHD and improvements in this lifestyle can lead to significant decreases in mortality statistics due to CHD (Cooper et al. 1978; Walker, 1983; Goldman and Cook, 1984). Goldman and Cook (1984) attributed greater than 50% of the reduction in mortality rate in U.S. from ischemic heart disease to improvements in lifestyle. During the period 1968—1976 there was a decline of 21% in the death rate from CHD and this percentage was even greater in persons under the age of 44. This is an important point because it means that changes are occuring in the younger people, and that influencing these younger adults can lead to dramatic results. As noted in the current study, the majority of the subjects

were under the age of 40, so it is vital to take the opportunity during the fitness evaluation to stress the importance of a healthy lifestyle.

Improved lifestyle through decreasing dietary fats, cessation of smoking and increasing physical activity all can significantly affect CHD risk factors (Wiley and Camacho, 1980; Taylor, 1983; Doll, 1983; Kannel and Schatzkin 1984). Dietary modifications lead to lowering of serum cholesterol and reductions in obesity. Physical activity plays a great role in reduction of serum cholesterol, obesity and blood pressure, and increasing serum HDL levels (Doll, 1983; Taylor 1983). Kornitzer (1984) recommends a lifestyle of maintenance of ideal body weight, regular exercise, reduction in dietary fat, avoidance of cigarette smoking and moderate alcohol intake.

Among the many roles of the fitness counsellor is the requirement to make people aware of these problems and their solutions. When a person has come to be fitness tested they are interested in their health or they would not be there. They are concerned about these important issues and it is an excellent moment which the counsellor must seize upon to influence the subject's lifestyle. This can be done through individual counselling and general education of the public at large. As previously mentioned greater than 50% of the population are not aware of the benefits of exercise and weight control, so it is very likely that they know even less about the relationships of exercise to these other risk factors (Fitness and Amateur Sport #23,1984). This is a great opportunity to make some changes and the appraiser/counsellor must be prepared to direct these changes.

The Canada Fitness Survey indicated that people generally behave according to what they perceive to be important and what they find to be fun (Fitness and Amateur Sport,1983). Therefore, it is crucial to point out the importance of a healthy lifestyle and the benefits of exercise. Equally as important is to make the experience of exercise participation enjoyable.

The Canada Fitness Survey also demonstrated that Canadians perceive 'feeling better', weight control and stress reduction as greater contributors to one's well-being than physical activity (Fitness and Amateur Sport,1983). The fitness counselling session is an enormous

opportunity to link all of these things together, because physical activity can contribute to weight control and stress reduction and ultimately to a sense of 'feeling better'. It is imperative that the emphasis on activity will reinforce good health practices.

Badura (1984) stated that "no one can deny that the excessive consumption of alcohol and nicotine, overeating and faulty nutrition, the lack of exercise and the misuse of drugs have considerable influence on well being and health status". However, he pointed out that it takes more than just stating the facts to influence people to change. Pressure must be applied to interested persons during their fitness testing sessions, through mass media, peer group pressure, social stress and working environment (Lawson and Blatch, 1981; Badura, 1984). Many of the lifestyle habits are formed through adolescence and young adulthood (Badura, 1984). The younger persons in the current study may then be more easily influenced to make changes in their lifestyle.

Shephard et al. (1982a and b) demonstrated the benefits of employee fitness programmes both financially, and in terms of one's health. The program involved regular fitness testing, physical activity, educational classes and individual correspondence through newsletters and mail-listings. They noted significant reductions in health risk when assessed through the health hazard appraisal questionnaire. The improvements were in the areas of increased activity, decreased smoking, decreased alcohol consumption and decreased blood pressure. They estimated the savings in total health care costs to be $84,500 per year for a company of 1,000 employees. This far outweighs the costs of the employee fitness programme itself.

In another paper, Shephard (1983) estimated that health care costs could be reduced by $45 million per year in the U.S. if all people between the ages of 20—69 were of average fitness. The costs for the fitness facilities and instructors would in no way equal this substantial reduction in health care costs. Additional benefits of the employee fitness/lifestyle program were worker satisfaction, improved lifestyle, decreased costs of cardiovascular disease, cigarette and alcohol related disease, decreased costs of physician services, increased productivity and

decreased absenteeism. Fitness counsellors can encourage persons to become involved in employee fitness programmes where such programmes exist.

Forty percent of women are members of the work force and therefore, subjected to the same stresses as their male counterparts (Hall and Richardson,1982). In addition, more women are smoking so that diseases such as CHD, which are prominent in this lifestyle will and are affecting more women and more often. Therefore, women require the same attention to fitness and healthy lifestyle as men. Perhaps the solution is to encourage working women to become involved with an employee fitness programme, and the non-working women to participate in less structured, time restrictive activities.

The 1983 data from the Canada Fitness Survey reported that only 6% of Canadians would become active after receiving information on the benefits of physical activity. An additional 9% said that having a fitness test would influence them to become active (Fitness and Amateur Sport,1983). In the current context of fitness testing, fitness counsellors should be able to positively modify the behaviour of 15% of Canadians. This is not that small a number, when considering the total Canadian population, and one does have to start somewhere.

Forty percent of people surveyed sited the 'lack of time' is as the major stumbling block to physical activity, but they would be active if this problem could be solved. Again, this is a great situation for the counsellor to help people find the time and priorize their activities.

There are great opportunities for fitness appraisers/counsellors and physical educators to play a role in modifying one's lifestyle into a more healthy state. This can be done through personal example, individual and group education sessions, public awareness programmes, employee fitness programmes to name a few.

LIMITATIONS

The major limitation in the current research undertaking is the validity of the original submaximal exercise test for cardiovascular fitness. This test was utilized to categorize the subjects into their fitness classification so that an invalid measure allows serious error to creep

into the experiment.

Each subject was given a classification based on the VO_2 max prediction from the treadmill test in addition to the original bicycle test. The same set of norms were used in each case but both VO_2 MAX values were predictions and not measured values. Thirty-eight percent of the subjects were re-classified into a different fitness level after the treadmill test. There was a definite regression towards the mean with the majority falling in the middle fitness level. Some changes would be expected due to the fact that the tests were only predictions; plus day to day individual variation in response exercise. However, 38% is a very large percentage and it makes the data difficult to interpret.

The correlation between the two VO_2 MAX predictions was .70, however the correlation between fitness classifications was only .59. As can be seen there was considerable error in these measures.

The reasons for these errors can be broken down into validity of the predictions and improper execution of the testing procedure. The validity of the cardiovascular tests (ie. Astrand bicycle ergometer test and the Bruce treadmill test) will now be discussed.

Validation of the Astrand bicycle test (Astrand and Rhyming, 1954) was carried out by comparing the prediction to direct measures of VO_2 MAX. Glassford et al. (1965) found a correlation coefficient of .79 between the bike test prediction and directly measured VO_2 MAX. deVries and Klafs (1965) found a correlation of .736 under the same circumstances. More recent work by Kasch (1984) found a correlation of only .58. He found the bike test underpredicted one's VO_2 MAX by 21%, and in up to 80% of people. These statistics point out the inherent errors in the cardiovascular test of fitness used to classify the subjects in the current investigation.

The correlation of .70 between the 2 tests used in this study is a correlation between 2 submaximal tests, both of which have sources of error. Therefore, the error for any one test may be greater than this value would suggest. Given this correlation, the best one could expect is to be accurate 50% of the time. No attempt was made to correlate the submaximal tests with

a maximal test; for this would have given a better indication of the error in the predictions.

The Bruce treadmill test (1971) also is submaximal and leads to a prediction of VO_2 MAX. This protocol was designed with cardiac patients in mind and not for fitness evaluation (Bruce et al. 1963). Despite this, a prediction of VO_2 MAX can be made based upon the total treadmill time in minutes (Bruce and Mc Donough, 1969; Bruce, 1971). This prediction of VO_2 MAX is determined through a regression equation based upon sex and total treadmill time as can be seen in table 21.

Table 21: Regression Equations and Correlation Coefficients for the Bruce Protocol (Bruce, 1971).

MEN

VO_2 MAX

= 8.38 + 2.94(treadmill time in minutes)

r = 0.936

WOMEN

VO_2 MAX

= 8.05 + 2.74(treadmill time in minutes)

r = 0.926

As can be seen above these correlation coefficients indicate a minimal amount of variability. This test is generally not used for predicting VO_2 MAX and validation research is not readily available. Even so this test is far from perfect and there is some error in its predictions as can be seen in Table 21. However, when using 2 tests with built-in error the potential for large amounts of error is even greater and again makes interpretation of the data difficult.

The validity of the two submaximal testing procedures has been dicussed. There are

three other points about the execution of these procedures that are important to mention. Firstly, some of the error in predicting VO_2 MAX may be due to failure of the tester to stress the cardiovascular system sufficiently. It is important to raise the heart rate high enough so that heart rate alone is responsible for the increases in cardiac output, and stroke volume no longer plays a role. This makes the relationship of heart rate to VO_2 MAX more predictable.

Stroke volume reaches a plateau and fails to increase further in the average person at 25—40% VO_2 MAX (Fox and Mathews, 1981; Brooks and Fahey, 1984). At work loads and heart rates below this level both stroke volume and heart rate contribute to increases in cardiac output, and therefore VO_2 MAX. If a prediction to VO_2 MAX is based on a heart rate, which is below the plateau level for stroke volume, the prediction will yield a higher than actual VO_2 MAX (ie. over prediction). The reason being, that if the changes in cardiac output were purely due to heart rate alone the heart rate would have to be higher to give that particular cardiac output. A higher heart rate for a particular work load means the person is less fit (ie. would have a lower VO_2 MAX). Therefore, it is important to elevate the heart rate high enough to eliminate the effects of stroke volume and make the prediction from the heart rate more accurate.

Secondly, measurement of the heart rate accurately is crucial to the accuracy of the prediction. Heart rate measurement throughout the 11 month testing period was inconsistent due to a variety of measurement techniques. The heart rate was generally measured by instruments such as a sport-tester or exer-sensory, but when the instruments were not functioning, manual measurement was used. Manual heart rate measurement is difficult when the heart rate is rapid, and it is very easy to miss one or two beats. Missing a few beats over a 10 second time period is multiplied by 6 when it is taken over a minute. Such miscalculations can lead to improper predictions for VO_2 MAX and thus wrongly classifying a person as high or low fit. For example, missing 3 beats in a 10 second period will lower the heart rate by 18 beats/min. This can result in a person being classified in the high fit group when they should have been in the low fit group. Jette et al. (1976) reported a correlation coefficient of .587

between subject palpation heart rates and ECG heart rates. However, with training this can be improved to a correlation coefficient of .94. Shephard et al. (1976) demonstrated that heart rates measured with an ECG were 7 beats/min higher than manually measured heart rates. As one can see there is considerable room for error in the prediction of VO_2 MAX based on manual heart rate monitoring.

Thirdly, the 2 testing procedures were different (ie. bike vs. treadmill). The bike test was a weight supported procedure whereas the treadmill was weight dependant. This would affect the VO_2MAX predictions, especially in the obese, unfit subjects; for walking on the treadmill would require more energy and place a greater stress on the cardiovascular system than riding a bike. This could help to further explain some of the discrepancy between the two VO_2MAX predictions.

The end result of this discussion is that the initial procedure is not without error so every attempt must be made to prevent further errors. Also, when the classification of subjects is a central part of the investigation another method of evaluating cardiorespiratory fitness should be considered.

Chapter V

CONCLUSIONS

The purpose of this study was to investigate the relationship between the levels of fitness, as determined in a university or community based testing fitness facility, to the risk factors for coronary heart disease in adult Canadians. The following statistically significant relationships were uncovered:

1. The unfit subjects were older than the fit subjects.

2. Proportionately more of the unfit persons illustrated elevated serum triglycerides than the fit persons.

3. The unfit persons possessed higher values for serum triglycerides, cholesterol, lipid ratios and fasting blood sugar, plus lower HDL values than the fit persons.

4. The unfit persons demonstrated higher resting systolic and diastolic blood pressures than the fit persons.

5. Consideration should be given to the use of 85 mmHg as the cut-off point for normal diastolic blood pressure by all fitness appraisal centres as well as all health professionals.

6. The unfit persons exhibited a greater likelihood of having a positive family history for CHD and diabetes mellitus.

7. The unfit persons displayed higher fasting blood sugar values than the fit persons.

8. The unfit persons had a greater percent of body fat and greater body weight than the fit persons.

9. The unfit persons consumed more alcohol per day than the fit persons.

10. The unfit are at significantly greater risk of developing CHD when multiple risk factors are considered.

This evidence points to one of an unhealthy lifestyle and it is essential for fitness counsellors to be aware of these problems when advising clients about fitness and lifestyle changes. The task of altering a person's lifestyle is a monumental one as the following quote

illustrates.

> Men as a rule find it easier to depend on healers than to attempt the more difficult task of living wisely.
>
> (Dubos, 1961)

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APPENDICES

APPENDIX A: PAR-Q AND CONSENT FORM

THE UNIVERSITY OF ALBERTA
FITNESS APPRAISAL SCREENING QUESTIONNAIRE

The questionnaire and consent form is designed to assist in identifying the small number of adults from whom this submaximal fitness test might be inappropriate. Please complete all parts of the questionnaire and sign it.

NAME: _____ ADDRESS: _____

Physical Activity Readiness Questionnaire* (PAR-Q) YES ( ) NO ( )

Has your doctor ever said you have heart trouble?

Do you frequently suffer from pains in your heart or chest?

Do you often feel faint or have spells of severe dizziness?

Has a doctor ever said your blood pressure was too high?

Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?

Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?

Are you over age 69 and not accustomed to vigorous exercise?

Consent for the Standardized Test of Fitness

I, _____, authorize the University of Alberta to administer and conduct an exercise fitness test designed to determine my physical work capacity. I understand that I will perform tests of grip strength, sit-ups, push-ups and trunk flexion.

I understand that I will pedal a bicycle ergometer for eight minutes at a resistance identified for my sex and age group. During the performance of the test my heart rate will be monitored and my blood pressure will be measured prior to, during, and at the completion of the test. The test will be discontinued when I reach a predetermined heart rate or if I become distressed in any way or develop any abnormal response which ever of the above occurs first. Every effort will be made to conduct the test in such a way as to minimize discomfort and risk. However, I understand that just as with other types of fitness tests there are potential risks. These include episodes of transient lightheadedness, fainting, chest discomfort, leg cramps and nausea.

I understand that I can discontinue the test at any time or choose not to perform specific items. The test has been fully described to me and I have had the opportunity to satisfy my questions about the tests.

In agreeing to such an examination, I waive any legal recourse against the members of the staff of the University of Alberta from any and all claims resulting from personal injuries sustained or death resulting from these tests. This waiver shall be binding upon my heirs and my personal representatives.

DATE: _____ SIGNATURE: _____

APPENDIX B: FITNESS UNIT DATA SHEET

UNIVERSITY OF ALBERTA
FITNESS TEST

NAME: _____ DATE OF TEST: _____

AGE: _____ SEX: _____

ANTHROPOMETRIC DATA

HEIGHT: _____ WEIGHT: _____

GIRTHS: Chest _____ Gluteal _____

Abdomen _____ Thigh _____

FINAL _____

SKINFOLDS:

TRICEPS	_____	_____	_____	_____	
SUBSCAPULAR	_____	_____	_____	_____	
BICEPS	_____	_____	_____	_____	
SUPRAILIAC	_____	_____	_____	_____	
				TOTAL	_____

BICYCLE ERGOMETER TEST

RESTING HR: _____ RESTING BP: _____

STAGE 1: Workload _____kpm
4 Min HR _____ BP: _____

STAGE 2: Workload _____kpm
7 Min HR _____ BP: _____
8 Min HR _____ BP: _____

POST EX HR (:30-1:00) _____ POST EX BP (:30-1:00) _____
POST EX HR (2:30-3:00) _____ POST EX BP (2:30-3:00) _____

MUSCULAR STRENGTH AND ENDURANCE, FLEXIBILITY TEST

GRID STRENGTH RT. _____ LT. _____ TOTAL _____

TRUNK FLEXION _____ cm

60 SEC. SIT-UPS _____

PUSH UPS _____

APPENDIX C: HEALTH HAZARD APPRAISAL QUESTIONNAIRE

HEALTH HAZARD APPRAISAL

Please answer all of the questions by marking the appropriate answer with a ( ), unless not applicable.

1. NAME_____
   ADDRESS_____
   _____
   POSTAL CODE _____
   PHONE NUMBER(H)_____(W)_____
   OCCUPATION_____

2. SEX ( ) 1...Male
   ( ) 2...Female

3. Height: (inches or centimeters)_____

4. Age_____

5. Weight: (pounds or kilograms)_____

6. Do you know your blood pressure? If so Systolic (Higher value)_____
   Diastolic (Lower value)_____

7. Do you know your cholesterol level? If so:
   Millimeters per deciliter (140-320)_____
   or International Units-millimoles per liter (3.6-8.3)_____

8. Activity level: ( ) 1...Little or none
   ( ) 2...Some activity
   ( ) 3...Moderate Exercise
   ( ) 4...Vigorous Exercise

9. Smoking Habits: ( ) 1...Smoker
   ( ) 2...Former Smoker
   ( ) 3...Never Smoked
   Which is (was) smoked more? ( ) 1...Cigarettes
   ( ) 2...Cigars and/or pipes
   If pipe: amount smoked per day ( ) 1...5 or more, or any inhaled
   ( ) 2...Less than 5, none inhaled
   If cigarettes: amount smoked per day ( ) 1...2+ packs
   ( ) 2...1 pack
   ( ) 3...Half pack
   ( ) 4...Less than half pack
   If quit, how many years since quitting? (If less than 1 year put 1)_____

10. Alcohol consumption: (drinks per week - apertifs, wine, beer, etc).
    ( ) 1...41 or more
    ( ) 2...25-40
    ( ) 3... 7-24
    ( ) 4...3-6
    ( ) 5...1-2
    ( ) 6...Quit
    ( ) 7...Never drank

11. How often do you wear a seatbelt while travelling in a car?
    Percent of the time _____.

12. Annual distance travelled in a car or on a motorcycle: (miles or kilometers) _____

13. Diabetes ( ) 1...yes-controlled
( ) 2...yes-uncontrolled
( ) 3...no

14. Family history of diabetes (parent or sibling) ( ) 1...Yes
( ) 2...No

15. Family history of ischaemic heart disease:
( ) 1...both parents died of a heart attack before age 60
( ) 2...one parent died of a heart attack before age 60
( ) 3...both parents lived to age 60
( ) 4...none of the above

16. Emphysema ( ) 1...signs or symptoms
( ) 2...no signs or symptoms

17. Bacterial Pneumonia ( ) 1...have had
( ) 2...have not had

18. Disabling Depression ( ) 1...often
( ) 2...seldom or never

19. Family history of suicide ( ) 1...yes
( ) 2...no

20. Rectal growth (other than hemorrhoids) ( ) 1...have had
( ) 2...have not had

21. Rectal bleeding (undiagnosed) ( ) 1...have had
( ) 2...have not had

FEMALES ONLY
CERVICAL AND BREAST CANCER RISK

22. Age regular intercourse began
( ) 1...under 20 ( ) 2...20-25 ( ) 3...over 25 or never

23. Pap smear: Have you had a hysterectomy? (Uterus or Cervix)
( ) 1...yes
( ) 2...no
If not, have you had a pap smear within 5 years? ( ) 1...yes
( ) 2...no
Was your most recent pap smear negative (ok)? ( ) 1...yes
( ) 2...no
Have you had 3 or more pap smears within 5 years, of which the last 3 were negative (ok)? ( ) 1...yes
( ) 2...no

24. Family history of breast cancer (mother or sister) ( ) 1...yes
( ) 2...no

25. Monthly breast self-examination ( ) 1...yes ( ) 2...no

APPENDIX D: ASTRAND-RHYMING NOMOGRAM

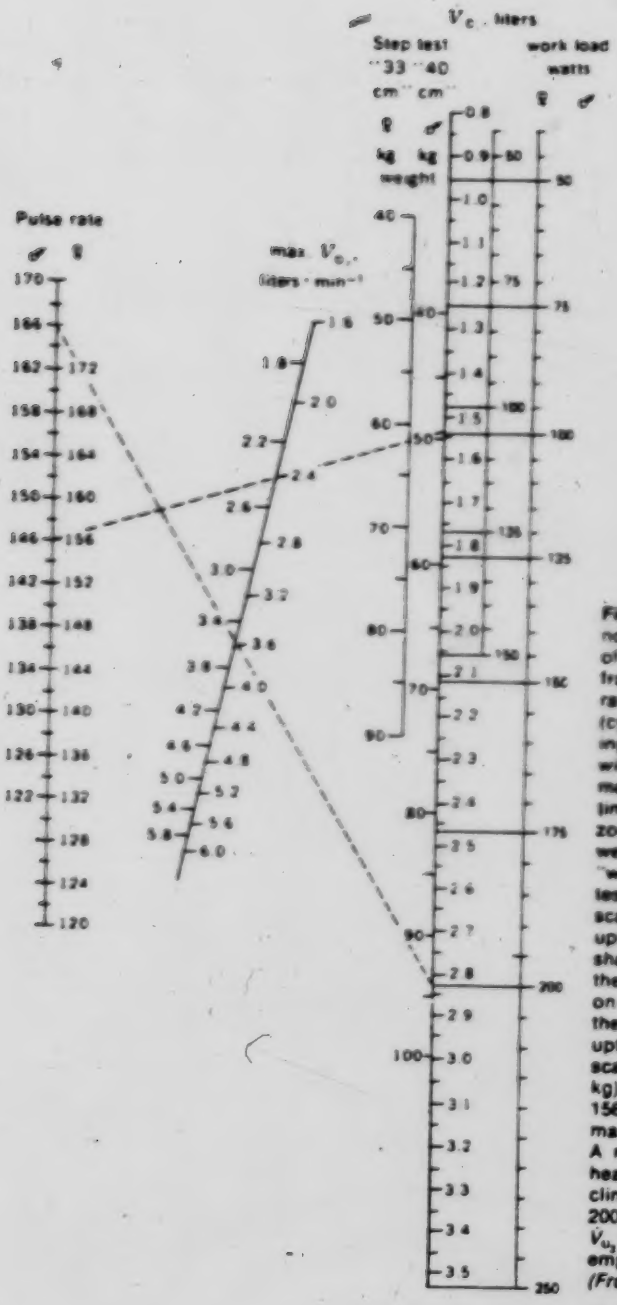

Figure 10-7 The adjusted nomogram for calculation of maximal oxygen uptake from submaximal pulse rate and O_2-uptake values (cycling, running or walking, and step test). In tests without direct O_2-uptake measurement, it can be estimated by reading horizontally from the "body weight" scale (step test) or "work load" scale (cycle test) to the "O_2 uptake" scale. The point on the O_2-uptake scale (V_{O_2} liters) shall be connected with the corresponding point on the pulse rate scale, and the predicted maximal O_2 uptake read on the middle scale. A female subject (61 kg) reaches a heart rate of 156 at step test; predicted max. $\dot{V}_{O_2}$ = 2.4 liters · min⁻¹. A male subject reaches a heart rate of 166 at cycling test on a work load of 200 watts; predicted max. $\dot{V}_{O_2}$ = 3.6 liters · min⁻¹ (exemplified by dotted lines). *(From I. Åstrand, 1960.)*

APPENDIX E: AGE CORRECTION FACTOR FOR ASTRAND-RHYMING NOMOGRAM

Table 6 a and b. Factor to be used for correction of predicted maximal oxygen uptake: a) when the subject is over 30—35 years of age *or* b) when the subject's maximal heart rate is known. The actual factor should be multiplied by the value that is obtained from Table 3 or Table 4.

Age	Factor	Max. heart rate	Factor
15	1.10	210	1.12
25	1.00	200	1.00
35	0.87	190	0.93
40	0.83	180	0.83
45	0.78	170	0.75
50	0.75	160	0.69
55	0.71	150	0.64
60	0.68		
65	0.65		

APPENDIX F: SWEDISH NORMS FOR CLASSIFICATION OF VO_2 MAX.

Table 7. Classification of Maximal Oxygen Uptake (maximal aerobic power) by Age Group. The upper figure, e.g. 1.69, refers to maximal oxygen uptake in l/min., the lower, e.g. 28, refers to ml/kg×min. "Normal weights" used were: 58 kg for females and 72 kg for males. (Ref. 2.).

Age	Maximal oxygen uptake, Vo_2 l. ml/kg×min.				
	low	somewhat low	average	high	very high
♀ 20–29	1.69	1.70–1.99	2.00–2.49	2.50–2.79	2.80
	28	29–34	35–43	44–48	49
30–39	1.59	1.60–1.89	1.90–2.39	2.40–2.69	2.70
	27	28–33	34–41	42–47	48
40–49	1.49	1.50–1.79	1.80–2.29	2.30–2.59	2.60
	25	26–31	32–40	41–45	46
50–65	1.29	1.30–1.59	1.60–2.09	2.10–2.39	2.40
	21	22–28	29–36	37–41	42
♂ 20–29	2.79	2.80–3.09	3.10–3.69	3.70–3.99	4.00
	38	39–43	44–51	52–56	57
30–39	2.49	2.50–2.79	2.80–3.39	3.40–3.69	3.70
	34	35–39	40–47	48–51	52
40–49	2.19	2.20–2.49	2.50–3.09	3.10–3.39	3.40
	30	31–35	36–43	44–47	48
50–59	1.89	1.90–2.19	2.20–2.79	2.80–3.09	3.10
	25	26–31	32–39	40–43	44
60–69	1.59	1.60–1.89	1.90–2.49	2.50–2.79	2.80
	21	22–26	27–35	36–39	40